

Battle Casualties in Korea

Studies of the
Surgical Research Team

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Battle Casualties in Korea:

Studies of the Surgical Research Team

Volume III. The Battle Wound: Clinical Experiences

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Preface

The third volume on experiences of the Surgical Research Team of Korea embodies the studies carried out in the clinical management of war casualties. This volume is divided into three parts. Part One represents the general aspects of surgical care. Part Two includes the management of specific clinical problems, such as vascular trauma, burns, débridement, and Clostridial myositis. Part Three reviews certain laboratory surveys of the clinical material, including bacteriologic studies, postmortem examinations, and an analysis of experiences with fat embolism.

The surveys and studies in the clinical care of patients were made at the 46th Surgical Hospital. This hospital was located on the eastern front 10 miles behind the main line of resistance. It supported the 45th Infantry Division, the 40th Infantry Division, and the Philippine Expeditionary Forces. It received casualties from such memorable battles as the Punch Bowl, Sandbag Castle, Heartbreak Ridge, and Christmas Hill. During the last 7 months of the war, it remained in the same position. The hospital was constructed of several tropical shells fastened together; and it had a concrete floor. Most of the hospital was lined with plywood. Several views of the interior of the hospital are shown in Figures 1 through 4. The patients arrived by ambulance and by small helicopter (Fig. 5). They were transported by ambulance to an airstrip about 10 miles away from the Surgical Hospital, and then flown to evacuation hospitals. Sometimes they were flown directly from the Surgical Hospital by large cabin-type helicopters (Fig. 6).

The clinical studies reflect experiences of the last 18 months of the Korean conflict. Captain John M. Howard, of the Surgical Research Team, and Captain Frank K. Iuni, Chief of the Surgical Service at the 46th Mobile Army Surgical Hospital, conducted many of these studies in 1952. Major Curtis P. Artz and Captain Yoshio Sako of the Surgical Research Team, and Captain Alvin W. Bronwell, Chief of the Surgical Service of the 46th Surgical Hospital, completed the studies in 1953. The results were compiled from operating room records and from statistical data sheets. The statistical data sheets were prepared as study sheets and incorporated, on admission, as part of the patient's chart. The surgeon responsible for a patient's care completed these sheets at the time he was evacuated; then they were kept on file with the Surgical Research Team.

The statistical data sheets gave rather extensive data on the care of each casualty. This method of maintaining records provided an opportunity for compiling the experiences of the hospital each month.

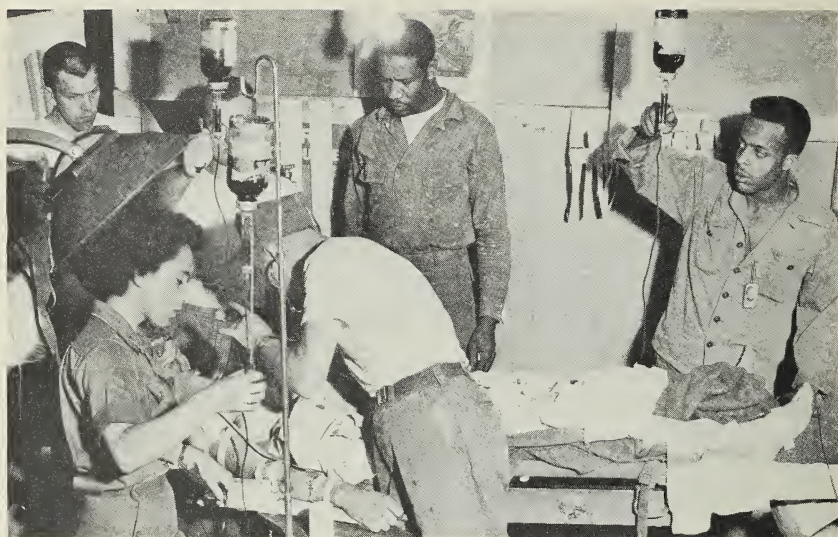


FIGURE 1. Scene in preoperative section of the 46th Surgical Hospital. This severely injured casualty is receiving blood rapidly through three intravenous portals. A surgeon member of the Surgical Research Team, the Chief of the Surgical Service, an anesthesiologist, and a staff surgeon are assisting in his care.



FIGURE 2. The X-ray section of the forward surgical hospital. Excellent facilities were available for roentgenographic examination.



FIGURE 3. The operating room of the 46th Surgical Hospital on a busy afternoon. In the foreground, two surgeons are performing a minor débridement. They do not wear operating room gowns. At the adjoining table, a major abdominal operation is under way. Lighting facilities were excellent.



FIGURE 4. Postoperative section. Immediately after operation, the patient's litter was placed on a cot. As soon as he reacted, he was moved to an iron bed farther back in the ward.

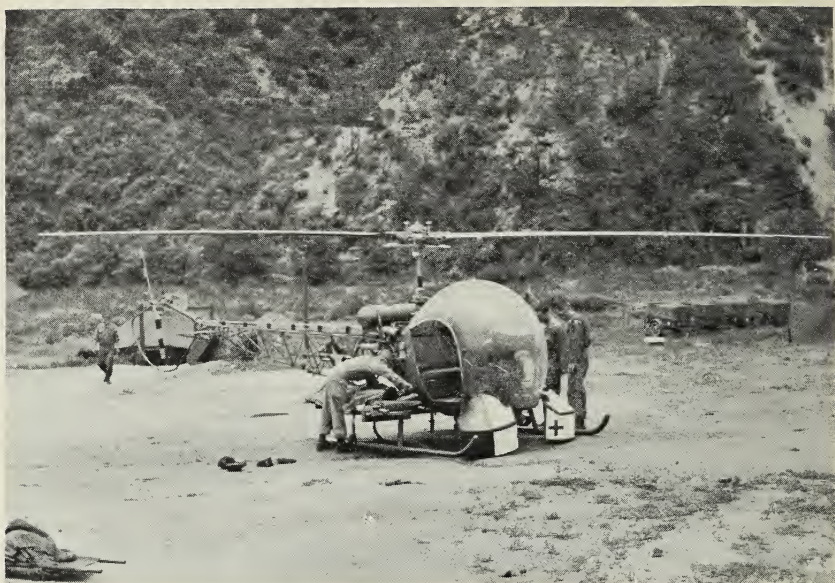


FIGURE 5. A small helicopter. This type of aircraft was used to bring selected casualties from the battalion aid stations to the surgical hospital. The helicopter landed only a few feet from the door of the admission section. Patients were then transported immediately by litter carry to the preoperative section.

The results were discussed at monthly surgical meetings, thereby giving the surgeons at the hospital an opportunity to review their experiences. This constant study of the care of large numbers of casualties resulted in "on-the-spot" improvement in management.

Most of the studies were carried out at the 46th Mobile Army Surgical Hospital in Korea, with the laboratory aspects being conducted at the 406th General Laboratory in Tokyo. Captain David B. Lester, MC, and 1st Lieutenants Faust Durden, MC, Richard L. Jankowska, MC, Walter Lawrence, Jr., MC, Robert C. Smith, MC, Donald L. Weeks, Jr., MC, and Joel W. Williamson, MC, members of the surgical staff of the 46th Surgical Hospital, participated in the evaluation of the material compiled from the statistical data sheets.

The investigations concerning arterial injuries were initiated by Major Edward J. Jahnke, Jr., MC, at the 43d Mobile Surgical Hospital in Korea during 1952; and data were then completed by Lieutenant Colonel Carl W. Hughes, MC, at the same hospital during the last 8 months of the war.

The Surgical Research Team took a very active part in coordinating the work at the hospital with the care at the battalion aid stations. Many battalion surgeons returned to the hospital frequently to discuss problems. Likewise, members of the Surgical Research Team



FIGURE 6. A large helicopter. This type of aircraft was used to transport patients between forward hospitals and from a forward hospital to the rear. Six litter patients could be carried at one time. This large helicopter was especially valuable when the casualty load at one hospital was too heavy for its facilities and several casualties had to be transported to an adjoining hospital.

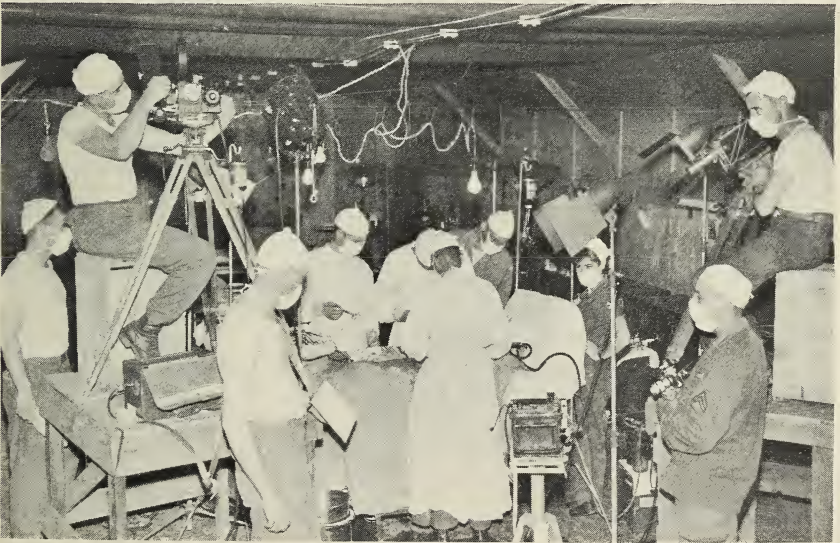


FIGURE 7. The medical photographic team in operation. Two motion picture cameras were used to cover the operative field, one for long shots, the other for closeups. In addition, several still cameras were available to record significant findings. This team of photographers was available only during the last month of the war.

visited the battalion aid stations, and the free interchange of ideas contributed to improvement in the care of casualties.

In keeping with the aim that these studies of the Surgical Research Team of Korea would provide a basis for teaching the technics of care of large numbers of casualties, a staff of movie photographers was sent to the 46th Mobile Army Surgical Hospital during the last month of the war. Under the direction of First Lieutenant Stephen P. Dittmann of the Army Medical Service Graduate School, this group took over 8,000 feet of colored movies on initial care of the casualty and of procedures in the operating room. This footage was obtained for the purpose of preparing training films in the management of severely injured casualties (Fig. 7). Several hundred still photographs in color were also taken in order to assist in the teaching of the management of war casualties.

From the material contained in this volume, considerable information may be gained from practical clinical studies conducted during the time of combat. These studies proved to be an exceptional aid in improving patient care in Korea. They served as a guide for changes of policy in the care of casualties; and they also increased the efficiency of the Medical Department in time of combat. It must be emphasized that several members of the Surgical Research Team worked in the hospital as operating surgeons and they assumed the entire responsibility for the care of patients under study.

In any future conflict, it would seem imperative to assign a group of surgeons interested in clinical surgery and research to a forward installation to study casualties. They should be provided with the research equipment and an additional staff whose primary duty would be to carry out investigations to aid in improving the care of the wounded soldier.

Fort Sam Houston, Texas
June 1, 1955

CURTIS P. ARTZ
Lieutenant Colonel, MC, USA



Part One

General Aspects of Surgical Care

Chapter 1

A Survey of Evacuation, Resuscitation, and Mortality in a Forward Surgical Hospital*

Captain Yoshio Sako, MC, USAR

Lieutenant Colonel Curtis P. Artz, MC, USA

Captain John M. Howard, MC, USAR

Captain Alvin W. Bronwell, MC, USAR

Captain Frank K. Inui, MC

Although more effective weapons have been devised with each succeeding war, the case fatality rate of the wounded soldier has steadily declined. An evaluation of the methods of treatment of the battle casualty requires accurate and complete recording of data concerning patient care, complications, and case fatality rate. These data may then be compared with data obtained from similar groups in previous wars. The dissemination of such information is a very important factor in the continued improvement in the treatment of battle casualties.

This report covers the period from January 1, 1952, to August 1, 1953,** of the Korean conflict, during which time the Surgical Research Team was active in studying various problems at the 46th Surgical Hospital (Mobile Army). This hospital was located approximately 10 miles behind the battle lines on the eastern front. The building was of tropical-shell construction and had a concrete floor. The hospital was equipped and staffed to do all types of major surgical procedures.

During the period covered by this report, large numbers of projects were being carried on and, as various ones were completed, others were initiated. In some instances, new projects were begun when there was a change in personnel. One of the last projects to be initiated was the survey of evacuation, resuscitation, complications, and mortality on every casualty admitted to the general surgical service.

Section I. Evacuation and Resuscitation of Casualties During the Final Three Months of the Korean Conflict

In this particular survey, the period covered was from May 1, 1953, to August 1, 1953. Although this represents a relatively short period

*Previously published in *Surgery* 37: 602, 1955.

**Records were lost for the month of January 1953.

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of time, some of the information gathered appears to reflect experiences gained in the previous 15 months. The survey was made when there was little change in the tactical situation, patient load, mode of evacuation, and when there was no change in the site of the hospital.

Study sheets covering statistical data were completed for all general surgical patients. These sheets were made an integral part of the patient's record while he was in the hospital; and they were removed only after the patient was evacuated to the rear or discharged to return to duty. The admitting officer completed all pertinent administrative data, and additional information on the patient's care from injury to discharge from the hospital was completed by the medical officer assigned to the particular patient. The statistical-data sheets were tabulated each month, and the staff utilized this information in discussions. Patients included in the survey were those sustaining wounds of the abdomen, chest, or combined wounds of the chest and abdomen, those requiring amputation, and those having open fractures.

Table 1 shows the total number of casualties and the mode of evacuation. There were 250 patients in this group; 75 had abdominal wounds, 29 thoraco-abdominal wounds, and 33 thoracic wounds. There were 31 patients who required amputations; and 82 sustained open fractures. Except for the group sustaining open fractures, approximately one-half of this group was evacuated by helicopter.

Table 1. Case Fatality Rates and Mode of Evacuation

Type of Wound	Total	Death	Mortality (per cent)	Ambu- lance	Helicopter	Not Stated
Abdominal	75	9	12.0	40	27	8
Thoraco-abdominal	29	3	10.3	19	8	2
Thoracic	33	2	6.1	17	10	6
Amputations	31	3	9.7	16	13	2
Open fractures	82	1	1.2	53	18	11
Total	250	17	-----	145	76	29

Time Intervals

Table 2 shows the average time interval for various phases of care. In the five groups, the average period from injury to admission was 3.6 hours, and from injury to surgery 6.6 hours. The operative procedure averaged 1.8 hours. The length of the operation for casualties with abdominal and thoraco-abdominal wounds was greater than for casualties with other types of wounds.

Table 2. Time Intervals

(Average in Hours)

Type of Wound	Number of Cases	Injury to Admission	Admission to Surgery	Injury to Surgery	Operative Time
Abdominal	75	3. 1	3. 2	6. 3	2. 4
Thoraco-abdominal	29	4. 7	2. 7	7. 9	2. 3
Thoracic	33	3. 7	3. 8	7. 5	1. 6
Amputations	31	4. 2	2. 6	6. 8	1. 6
Open fractures	82	3. 5	2. 7	6. 2	1. 3
Total Average	250	3. 6	3. 0	6. 6	1. 8

Resuscitative Fluids

Table 3 lists the average amount of blood administered to the various types of casualties. Several in this group received 1 unit of dextran or albumin prior to admission; whereas only a few received blood prior to admission. All but the open fracture group were given an average of 2 liters of blood preoperatively. During operation, patients received an average of more than 2 liters of blood, except in the casualties sustaining chest wounds and those having open fractures.

In the first 24 hours following injury, patients having abdominal wounds and those requiring amputations were given an average of 3.5 liters of blood. Patients having thoraco-abdominal wounds, thoracic wounds, and open fractures, received an average of 2.9, 1.9, and 0.9 liters of blood respectively in the first 24-hour period.

Table 3. Blood Transfusions

(Average Cubic Centimeters Transfused)

Type of Wound	Number of Casualties	Preadmission	Preoperation	During Operation	1st 24 Hours After Opn.
Abdominal*	70	264	1, 464	1, 467	3, 428
Thoraco-abdominal	29	103	1, 308	1, 187	2, 867
Thoraco	33	75	1, 088	333	1, 890
Amputations**	30	469	1, 403	1, 407	3, 500
Open fractures	82	47	380	448	926

*These figures do not include casualties who were given 40, 46, 52, and 56 pints of blood respectively, and one casualty on whom no record was available.

**One casualty who received 35 pints of blood was not included.

Deaths and Case Fatality Rates

Table 4 lists the deaths and the case fatality rates for each group. There were nine deaths in the group having abdominal injuries, a case fatality rate of 12 per cent. Of this number, five patients died of uncontrolled hemorrhage, two of peritonitis, and one each of aspiration pneumonia and pancreatitis. There were three deaths in the thoraco-abdominal group, a case fatality rate of 10.3 per cent. The causes of death were uncontrolled hemorrhage, postoperative shock, and cardiac arrest. There were two deaths among patients suffering from thoracic wounds, a case fatality rate of 6.1 per cent. One patient died of a massive, uncontrolled hemorrhage from a lacerated pulmonary vein; and one patient died of air embolism which occurred during positive pressure transfusion. There were three deaths in patients suffering from traumatic amputations, a case fatality rate of 9.7 per cent. One patient died of uncontrolled hemorrhage, one of cardiac arrest following excessive morphine, and one of massive hemorrhage from the thigh immediately after admission to the hospital. In the open fracture group there was only one death, an incidence of 1.2 per cent. This patient died of uncontrolled hemorrhage from massive wounds of both upper thighs. He was given 12 pints of blood in 30 minutes, but failed to respond. In the casualties described above, more than half of the deaths were caused by uncontrolled hemorrhage.

Table 4. Causes of Death

Type of Wound	Number of Cases	Number of Deaths and Percentage	Causes of Death	Total
Abdominal	75	9 (12.0%)	Uncontrolled hemorrhage	5
			Peritonitis	1
			Aspiration pneumonia	1
			Pancreatitis	1
Thoraco-abdominal	29	3 (10.3%)	Uncontrolled hemorrhage	1
			Postoperative shock	1
			Cardiac arrest	1
Thoracic	33	2 (6.1%)	Air embolism	1
			Massive hemorrhage	1
Amputations	32	3 (9.7%)	Uncontrolled hemorrhage	1
			Cardiac arrest after hypomorphenism	1
			Massive hemorrhage (died 10 min. after admission)	1
Open fractures	82	1 (1.2%)	Unknown (died 20 min. after admission; given 12 pts. blood)	1

Section II. Case Fatality Rates in 1952 and 1953

Data on total admissions were obtained for the period from January 1, 1952, to January 1, 1953. During this time, 3,716 general surgical patients were admitted to the 46th Surgical Hospital; and 83 of these patients died making a case fatality rate of 2.2 per cent. During the period from February 1, 1953, to August 1, 1953, 995 general surgical patients were admitted to the hospital; and the case fatality rate for these patients was 3.2 per cent. During the two periods mentioned, various types of patients were admitted. In 1952, all but a few casualties requiring operations were admitted to the hospital. In 1953, many of the less severely wounded soldiers were operated on at a clearing station. Over-all case fatality rate for these two periods was 2.4 per cent.

From January 1, 1952, to January 1, 1953, 327 patients were admitted to the hospital who had abdominal injuries; and 42 deaths occurred in the group, a case fatality of 12.8 per cent. From May 1 to August 1, 1953, 9 deaths occurred among the 75 casualties sustaining abdominal wounds, a case fatality rate of 12 per cent. The combined case fatality rate for these two groups was 12.6 per cent.

From January 23, 1952, to October 1952, and from February 1, 1953, to August 1, 1953, there were 140 casualties who required major amputations (Table 5). Of this number, 11 deaths occurred, a case fatality rate of 7.8 per cent. Table 5 shows the distribution of the levels of amputation and the number of deaths in each category.

Table 5. Major Amputations*
(January–October 1952 and February–August 1953)

Upper extremities	Total		Lower Extremities	Total	
	Number	Deaths		Number	Deaths
Above elbow	9	1	Above knee	16	3
Below elbow	27	1	Bilateral above knee	3	1
Bilateral below elbow	1	0	Below knee	64	3
Both Extremities			Bilateral below knee	7	0
			Bilateral above and below knee	8	1
Both legs & one arm	1	1	Total	140	11
Both legs & both arms	1	0			
Below knee and below elbow	3	0	Case Fatality Rates 7.85 per cent		

* Includes hands and feet, but not digits.

Section III. Comparison of Experiences in Korea with a Similar Group in World War II

It is difficult to make an accurate comparison of mortality statistics of any two wars, or even of various groups in the same war, because of the many factors contributing to the mortality in the wounded. Forsee stated that no sizable group of American military surgeons ever had such a vast experience in the surgical care of the severely wounded as did the Second Auxiliary Surgical Group in World War II.¹ This group worked in the first priority surgical hospitals whose location and function in the chain of evacuation was similar to that of a Mobile Army Surgical Hospital in Korea. For this reason, the findings of the Second Auxiliary Surgical Group were a comparable basis for evaluation of the statistical data gathered in Korea.

Evacuation Time

Table 6 shows a comparison between World War II and Korea of time intervals for evacuation of casualties with abdominal wounds. The time interval from injury to admission to the surgical hospital was shorter during the Korean conflict; but the interval from admission to surgery was not greatly changed. Usually, the interval from injury to admission was approximately twice as long during World War II as it was in Korea.

Table 6. Comparison of Time Intervals in Casualties with Abdominal Wounds

Time Interval	World War II, 1945* (average in hours)	Korean Con- flict, 1953 (average in hours)
Injury to admission	5. 6	3. 1
Admission to surgery	3. 4	3. 2
Injury to surgery	8. 9	6. 3

*Second Auxiliary Surgical Group.²

Resuscitative Fluids

Comparative data on resuscitative fluid is difficult to obtain for World War II. Table 7 shows the report of the Second Auxiliary Surgical Group on the use of blood and plasma administered to casualties who had colon and rectal wounds. This report mentioned that rarely was there any limit to the supply of blood and plasma. No time interval was stated for periods during which blood was given;

however, it is assumed that the interval includes all periods during which blood and plasma were given while a casualty was at the First Priority Hospital. The total amount of blood given to the casualties who survived approximated 2 liters of resuscitative fluids per patient. The casualties who died had received approximately 3.5 liters, while the average for both groups was 2.5 liters.

Table 7. Blood and Plasma Transfusions in Casualties with Colon and Rectal Injuries, World War II*

Condition of Casualty	Blood		Plasma	
	Number of Patients	Transfused (ave. in cc.)	Number of Patients	Transfused (ave. in cc.)
Living	721	1, 415	706	610
Dead	410	2, 590	400	888
Total	1, 131	1, 840	1, 106	728

*Second Auxiliary Surgical Group, World War II.

In World War II, casualties who had colon and rectal wounds received an average of about 1 liter less of resuscitative fluids than did the casualties who sustained abdominal wounds in the Korean conflict (Table 3).

Comparison of Case Fatality Rates

It is difficult to make an accurate comparison of case fatality rates for World War II and for those reported in this survey, because no comparable groups are reported. In the Mediterranean theater of operations from January to June 1944, the case fatality rate of 34,731 casualties was 3.3 per cent.² This group excluded those injured soldiers who were returned to duty from the battalion aid stations, clearing stations, and dispensaries. In this 18-month survey of the Korean conflict, the over-all case fatality rate was 2.4 per cent for the general surgical patients at the 46th Surgical Hospital.

Our study shows that among those who had abdominal injuries, the organ most frequently involved was the colon (Table 8). Second in frequency were injuries of the small intestine and liver. The next most commonly injured organs were the kidney, spleen, and stomach, in that order. Incidence of organ injury followed in order rather closely the figures compiled for World War II. During our series, the case fatality rates of all groups were consistently lower than in the World War II series; and, in many instances, the rate was approximately half that of World War II. An exception in our study was injury to duodenum; 17 patients had duodenal injuries, a case fatality

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rate of 43.1 per cent. Our survey indicated a high rate of mortality in patients with ureteral injuries. In four such patients, the case fatality rate was 50 per cent; whereas, among 27 cases of ureteral injuries in the World War II series, it was 41 per cent.

Table 8. Comparative Statistics for Case Fatality Rates of Abdominal Wounds by Organs Injured—Korea, 1952–1953, and World War II, 1942–1945*

Viscus	KOREA				WORLD WAR II			
	Deaths, All Cases		Complicated† Per- centage Dying	Uncom- plicated Per- centage Dying	Deaths, All Cases*		Complicated† Per- centage Dying	Uncom- plicated Per- centage Dying
	Number	Per- centage			Number	Per- centage		
Colon	140	15.0	18.5	9.3	1,106	37	41	23
Jejunum and Ileum	134	13.4	16.4	3.0	1,168	30	36	14
Liver	102	15.6	20.6	9.0	829	27	39	10
Stomach	45	17.5	22.8	0	416	41	42	29
Kidney	55	25.4	29.1	0	427	35	38	16
Spleen	54	15.0	18.6	0	341	25	30	12
Rectum	22	18.1	16.0	18.7	155	30	42	14
Bladder	21	9.4	13.3	0	155	30	34	0
Duodenum	17	41.1	43.7	0	118	56	56	50
Pancreas	9	22.2	25.0	0	62	58	57	100
Gallbladder	33	0	0	0	53	30	30	0
Ureter	4	50.0	50.0	0	27	41	42	0

*Second Auxiliary Surgical Group.²

†Additional organ damage.

To equalize the time interval from injury to admission, the series covering the Korean conflict was grouped according to the number of organs injured in those casualties who were operated on within seven hours following injury (Table 9). This method was adopted in order to compare results with a similar group of casualties in World War II on which tabulation was made by Beebe and De Bakey. In their series of 1,185 casualties with abdominal wounds, the case fatality rate was 21 per cent; whereas, in the 384 casualties reported here, it was 11.9 per cent.² In the World War II series, the casualties included those who were operated upon *within 7 hours* following injury; whereas, in the Korean conflict series, the casualties included those operated upon *within an average of 7 hours* after injury. Consequently, in the latter group, many casualties were undoubtedly operated on at a time which exceeded 7 hours after injury. Since the time element was essentially comparable, the lower case fatality rate in the Korean conflict series cannot be ascribed entirely to the shorter period of evacuation.

Comparison of data on thoraco-abdominal wounds again revealed that the case fatality rate according to organ injured in our series

Evacuation, Resuscitation, and Mortality

Table 9. Comparative Statistics for Case Fatality Rates for Abdominal Wounds by Number of Organs Injured

Number of Abdominal Organs Hit	KOREA		WORLD WAR II		Statistical Tests for Significance of Differences CHI SQUARE analyzed according to the number of abdominal organs injured gives: $X^2 (5) = 11.95$ $0.05 > P > 0.02$ Over-all statistics $\frac{X}{S. D.} = 4.35$ $P < 0.003$
	Operation to Injury (aver. of 7 hrs.)		Operation to Injury* (7 hrs. or less)		
	Total Wounded	Per-centage Dying	Total Wounded	Per-centage Dying	
0	36	2. 80	98	5	
1	181	6. 62	496	10	
2	102	6. 82	402	24	
3	45	26. 60	132	42	
4	12	58. 30	41	54	
5	6	16. 00	13	92	
6	2	50. 00	3	100	
Total Average	384	10. 68	1, 185	20. 51	

*Second Auxiliary Surgical Group, World War II.²

Table 10. Case Fatality Rates of Thoraco-abdominal Wounds by Organs Injured

Organ	Korea 1952-1953			Second Auxiliary Surgical Group, 1942-1945		
	Total Wounded	Deaths		Total Wounded	Deaths	
		Number	Percentage		Number	Percentage
Liver	52	6	11.5	507	137	27
Spleen	25	4	16.0	279	80	29
Stomach	23	1	4.3	211	93	44
Large intestine	27	6	22.2	191	95	50
Small intestine	24	9	37.5	115	54	47
Kidney	17	5	29.4	121	71	59

was approximately half of that in the group studied by Beebe and De Bakey (Table 10). The greatest difference occurred in those casualties sustaining gastric wounds. In our study, 23 patients having gastric injuries had a case fatality rate of 4.3 per cent as compared with the World War II series in which there were 93 deaths among 211 casualties (44 per cent).

Further analysis of the thoraco-abdominal injury groups was made in terms of the side of the chest involved. In our series, 72 of the 129 thoraco-abdominal injuries were primarily on the right side and 57 on the left, with a case fatality rate of 16.6 and 8.7 per cent respectively (Table 11). In the World War II series, of 883 thoraco-abdominal injuries, the number was approximately equal on the right and left sides (435 on the right side, and 448 on the left). The case fatality rates in the latter series were approximately the same for both sides (24 per cent for the right and 30 per cent for the left).

Discussion

One of the important determining factors in the causation of death depends upon the body region which is injured and more particularly, the tissue involved. The study by Beebe and De Bakey of casualties in the Mediterranean Theater showed that, among the 36,000 wounded soldiers who had lost time because of injury during the first six months of 1944, the case fatality rate was higher in those suffering from abdominal injuries (17.4 per cent). Chest wounds stood second to abdominal wounds in case fatality rate (8.1 per cent), and head, face, and neck wounds were 5.7 per cent; the lower extremities, 2.1 per cent; and the upper extremities, 1.1 per cent.²

For a number of reasons, a missile wound of the chest, abdomen, buttocks, and upper thigh which was equally extensive, would probably have a decreasing mortality in the order listed. In all probability, an extensive wound of the chest would prove fatal in a very short time following injury; whereas, an equally large wound of the abdomen would probably injure multiple organs and bring about an equally high case fatality rate (*see* Table 9). In the regions of the buttocks and thigh, a wound would probably bring about a case fatality rate directly proportionate to the ease or difficulty of hemorrhage control.

When comparing regions of the body involved with case fatality rate, there appears to be a differential in the rates which have been unchanged during the different wars, even though the over-all rate has decreased. Case fatality rate may be related to blood loss because of injuries involving various regions of the body. However, it may be more closely related to the ease with which control of blood loss is brought about. For example, a simple laceration of an artery in the thigh is easier to control than in a wound where there is multiple extensive, soft-tissue damage in the same area.

In some areas of the body, a higher incidence of complications may be expected due to peculiarities of the specific region. In the abdomen, aside from blood loss, there is the grave complication of infection of the peritoneal cavity secondary to perforation of the gastro-

Table 11. Comparative Case Fatality Rates for Thoraco-abdominal Wounds

Conflict	Right Diaphragm			Left Diaphragm			Total		
	Number Wounded	Deaths		Number Wounded	Deaths		Number Wounded	Deaths	
		Number	Per Cent		Number	Per Cent			
Korea, 1952-1953	72	12	16.7	57	5	8.8	129	17	13.1
	435	103	24.0	448	136	30.0	883	239	27.0

intestinal tract. In the case of muscle wounds of the thighs and buttocks, there is the ever-present danger of myositis. In chest wounds, the location of the vital organs and of major vessels and pulmonary function which may be complicated postoperatively, contribute to a higher case fatality rate for injuries in this area.

Each war presents a different set of circumstances, and this may reflect upon case fatality rates. Rates would be higher in a fast-moving war where casualty loads would be heavy. An increased mortality rate would be reflected in this instance due to compromise of adequate preoperative and postoperative care because of the heavy patient load and the long hours of duty for surgeons, with subsequent loss of efficiency of personnel. On the other hand, in a long-drawn-out war, there may be better organization of the medical units, with improvement in patient care. Situations existing during the Korean conflict were almost ideal. The battle line was stable; control of the air was in friendly hands; and the hospital was situated close to the battle front. The casualty load was not excessive in one site; and the investigators were able to keep patients for a longer time in the postoperative period prior to evacuation. At no time was there need of compromising patient care because of lack of supplies or shortages of whole blood.

The lower mortality rate in the Korean conflict may be ascribed to a number of factors: the shorter evacuation time, the administration of large quantities of resuscitative fluids preoperatively and during surgery, and the routine use of antibiotics. Undoubtedly all of these factors contributed to a lower mortality rate. Although a shorter evacuation time permits the wounded to be treated soon after injury, a shorter evacuation time will tend to increase the over-all mortality rate because the more severely wounded casualties will reach the surgical hospital. Because of the availability of helicopters for evacuation during the Korean conflict, no doubt many patients were admitted to surgical hospitals who ordinarily would not have survived. Consequently more seriously wounded casualties were seen frequently in the forward surgical hospitals in Korea. The greatest single difference in the management of casualties in Korea appears to be the large quantities of blood administered throughout resuscitative periods. Beebe and De Bakey state that deaths from shock in the Fifth United States Army hospitals during World War II showed that, of 254 who died of shock following surgery, 210 (82 per cent) received no blood during or after surgery. Furthermore, 87 received no blood therapy prior to surgery.² In the deaths that occurred during the period covered by 3 months' survey, all patients who died postoperatively of either uncontrolled hemorrhage or shock had received more than five pints of blood in the first 24 hours following surgery.³

Summary

Results of detailed study of evacuation, resuscitation, and case fatality rates at the 46th Surgical Hospital in Korea from May 1, 1953, to August 1, 1953, have been reported. The average evacuation time from injury to admission was 3.6 hours and from admission to surgery 3.0 hours. Casualties with extremity wounds requiring amputations received an average of 3,500 cc. of blood in the first 24 hours following injury. Casualties with abdominal wounds, thoraco-abdominal wounds, thoracic wounds, and open fractures were given an average of 3,428 cc., 2,867 cc., 1,890 cc., and 926 cc., respectively, during the first 24 hours following injury. The highest mortality rates occurred in those casualties with abdominal wounds, nine deaths among 75 casualties (12 per cent); the lowest in those casualties with open fractures, one death among 82 casualties (1.2 per cent).

The over-all case fatality rate for 4,671 casualties admitted to the general surgical service over the period from January 1, 1952, to August 1, 1953, was 2.4 per cent. During this period, 402 casualties with abdominal wounds were admitted to the hospital. There were 51 deaths, a case fatality rate of 12.6 per cent.

The most important single factor in the improvement in mortality rates appears to be the greater amounts of blood given during resuscitation for our series than for those reported for World War II.

References

1. Forsee, James, H., Colonel, MC, USA: Forward Surgery of the Severely Wounded. Reprint from *The American Surgeon*, (Vol. XVII, 508-526), June 1951.
2. Beebe, G. W., and De Bakey, M. E.: Battle Casualties. Charles C Thomas, Springfield, Illinois, 1952.
3. Artz, C. P.; Howard, J. M.; Sako, Y.; Bronwell, A. W., and Prentice, T.: Clinical Experiences in the Early Management of the Most Severely Injured Battle Casualties. *Ann. Surg.* 141: 285, 1955 (Chapter 2 of this volume).

Chapter 2

Clinical Experiences in the Early Management of the Most Severely Injured Battle Casualties*

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War provides an exceptional experience from which the surgeon may gain information about the management of trauma. In the latter phases of the Korean conflict, the flow of casualties was moderate and the line of resistance was relatively stable. This resulted in an unusual situation whereby the forward hospital remained fixed for long periods and careful studies of the casualties could be carried out.

Selective helicopter evacuation permitted many critically wounded soldiers to reach the hospital alive who would have died during evacuation in fast-moving combat situations similar to those experienced in World War II. Since an extraordinarily large number of severely injured casualties were treated, it was believed wise to record data concerning their management.

A survey was carried out during the latter part of the Korean conflict at the 46th Surgical Hospital (Mobile Army) located approximately 10 miles behind the eastern front. All of the casualties in this survey were general surgical patients, excluding casualties having wounds of the brain and spinal cord. They were classified as the most severely wounded because they required replacement of at least one-half of their blood volume. This classification follows closely the classification used by Grant and Reeve in World War II.¹ The casualties were divided into several categories. Criterion for the various categories was the amount of blood required for resuscitation in the first 24 hours after injury, since most casualties are successfully resuscitated or die within this period. It was the belief that this classification might furnish a rough index of the degree of injury.

It is difficult to grade the degree of injury because there are various contributing factors, such as location of the wound, organs damaged, velocity of the missile, time lag, blood loss, and duration of hypovolemia. Some casualties sustaining head or heart wounds required

*Previously published in *Annals of Surgery* 141 : 285, 1955.

little blood; nevertheless they died. These wounded soldiers had vital organ damage and obviously were severely injured casualties. Since requirement of blood was not a primary factor in the resuscitation of soldiers sustaining head wounds, such casualties were excluded from the study. Most casualties sustaining heart wounds died before they reached the hospital.

Since most wounds are accompanied by hemorrhage of a significant degree, the severity of the injury might well be graded in part, at least, on the basis of blood loss. Blood loss begins with injury and continues throughout the preoperative, operative and, to a lesser extent, the postoperative phases of the patient's care. One gross indication of the total amount of blood lost appears to be the amount of blood required for successful resuscitation. Experience has demonstrated that restoration of the circulation to a stable state occurs within approximately 24 hours after injury. It appears that the total amount of blood required to resuscitate a casualty during the first 24 hours might be a reasonable indication of blood loss and, for clinical purposes, an indication of the severity of the injury.

This survey was prepared from available, dependable records; and it was divided into two parts. The first part was comprised of records of 138 casualties requiring from 5 to 56 pints of blood who were treated between February 1953 and August 1953. The second part consisted of records of 89 casualties treated between January 1952 and August 1953 who received 15 or more pints of blood or plasma expander. Casualties in the first group—those requiring 15 or more pints of blood or plasma expander—were also included in the second part of the survey.

One Hundred and Thirty-eight Consecutive Casualties Who Required Five or More Pints of Blood

Between February and August 1953, 995 battle casualties (684 American and 311 Korean soldiers) were admitted to the general surgical service. Only the most severely wounded Korean casualties were transferred to the United States Army Hospital and their evacuation time was prolonged. The case fatality rate for the American patients was 2.2 per cent and the over-all case fatality rate for all general surgical casualties was 3.2 per cent.

Of these 995 casualties, 138 (14 per cent) were classified as most severely wounded.

Case Fatality Rate and Blood Requirement

The case fatality rate in relation to blood requirement in this group of 138 patients is outlined in Table 1. As the blood requirement increased, mortality increased. Twenty casualties died and, of this

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**Table 1. Case Fatality Rate Related to Blood Requirement
(February to August 1953)**

Amount of Blood Given	Number of Patients	Deaths	Case Fatality Rate
5-10 pints	61	1	1.6%
10-15 pints	50	8	16.0%
15-20 pints	12	3	25.0%
20-56 pints	15*	8	53.3%
Total series	138†	20	14.5%

*Only 10 lived longer than 48 hours.

†Only 122 lived longer than 48 hours.

number, 16 died within the first 48 hours. Of the 122 patients who survived 48 hours, 6 developed a marked degree of renal insufficiency; and 4 of these 6 died.

Case Fatality Rate and Type of Injury

The case fatality rate in relation to the type of injury is outlined in Table 2. Casualties whose injuries were primarily in the abdomen had a case fatality rate of 16 per cent. In the group whose injuries were primarily in the extremities, the rate was 10 per cent. The casualties sustaining thoraco-abdominal wounds had a higher case fatality rate (26.6 per cent); for those who had chest wounds it was 9 per cent.

Table 2. Case Fatality Rate Related to Type of Injury

Type of Injury	Number of Patients	Deaths	Case Fatality Rate
Abdominal	60	10	16.0%
Extremity	52	5	10.0%
Thoraco-abdominal	15	4	26.6%
Chest	11	1	9.0%
Total series	138	20	14.5%

Evacuation to the Hospital

The type of evacuation to the surgical hospital from a division medical installation was carried out in accordance with the judgment of the battalion surgeon. When flying conditions were suitable, a helicopter was used if the surgeon felt that the patient could not with-

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stand transportation to a hospital by ambulance or if he felt that it was necessary to transport a casualty quickly. Available data on 99 of the 138 casualties showed an equal division of type of evacuation between helicopter and ambulance. Of those who required over 10 pints of blood, a slightly higher percentage was moved by helicopter.

Evacuation time, time in the preoperative section, and duration of operation are outlined in Table 3. The time from injury to admission was 3.8 hours for 117 patients on whom data were available. In the 5- to 10-pint group, evacuation time was only slightly longer (4.2 hours) than in the 10- to 56-pint group (3.5 hours).

Table 3. Evacuation Time and Time of Initial Care

Category	Patients Reported	Hours Injury to Admission	Hours Admission to Operation	Hours Injury to Operation	Hours Duration of Operation
5-10 pint group	54	4.2	3.6	7.8	2.0
10-56 pint group	63	3.5	3.3	6.8	2.9
Total Average	117	3.8	3.5	7.3	

An average of 3.5 hours was spent from the time of admission to the time of operation. This interval was almost the same for both the 5- to 10-pint and the 10- to 56-pint group. However, it was not necessarily the time required for preparation for operation. In most instances, patients were held in the preoperative section until ready for surgery. If a large number of casualties were admitted at the same time, some casualties who were fully prepared for surgery had to wait for available space in the operating room. Priority was given to severely wounded casualties who were taken to the operating room as soon as possible. Average time from injury to operation was approximately 7.25 hours.

Casualties in the 5- to 10-pint group required 2 hours for operation and the more severely wounded casualties required about 3 hours.

Resuscitative Fluids During Various Phases of Care

The amount of blood or colloid solution given prior to admission to the hospital is summarized in Table 4. Among the 138 patients, 81 received resuscitative fluids, predominantly dextran; but some received modified fluid gelatin, albumin, and blood. Patients in the 5- to 10-pint group received about 1 pint of blood or plasma expander; patients in the 10- to 56-pint group received about 3 pints of fluid prior to admission.

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Table 4. Preadmission Blood and Plasma Expander

Category	Total Number of Patients in Category	Number Receiving Therapy	Average Amount Received—Preadmission*
5-10 pint group	61	38	685 cc.
10-56 pint group	77	43	1,440 cc.
Entire series	138	81	1,090 cc.

*Mostly dextran.

The average amount of blood replacement during various phases of resuscitation is shown in Table 5. Patients in the 5- to 10-pint group were given an average of 3,440 cc. of fluid therapy during the first 24 hours after injury. Those requiring from 10 to 35 pints averaged more than twice as much as the first group (7,300 cc.). In each group, the amount of blood given preoperatively was essentially the same as the amount given during the operation. Out of the 73 patients in the 10- to 35-pint group, 38 received an average of 1,130 cc. postoperatively; and 18 of the 61 patients in the 5- to 10-pint group received 720 cc.

Table 5. Blood Replacement During Phases of Resuscitation

Category	Number Receiving Therapy†	Amount Prior to Operation	Amount During Operation	Amount Post-operative
5-10 pint group				
Total patients—61	59	1, 770 cc.		
Average total blood in first 24 hours—3,440 cc.	55		1, 500 cc.	
	18			720 cc.
10-35 pint group*				
Total patients—73	67	3, 400 cc.		
Average total blood in first 24 hours—7,300 cc.	66		3, 200 cc.	
	38			1, 130 cc.

*For more accurate sampling, 4 patients who received 40, 46, 52, and 56 pints of blood were excluded.

†Only those patients included on whom data were available.

Fifty per cent of the patients in the abdominal group and 48 per cent of the patients in the extremity group were given approximately 1 liter of plasma expander, in addition to blood (Table 6). Most of this plasma expander was administered before these patients arrived at the hospital.

Table 6. Amount of Plasma Expander in Addition to Blood*

Type of Injury	Number of Patients in Category	Number Receiving Plasma Expander	Average Amount of Plasma Expander
Abdominal	60	30 (50%)	1,050 cc.
Extremity	52	25 (48%)	922 cc.
Thoraco-abd.	15	----- not significant -----	
Chest	11	----- not significant -----	

*Mostly given preadmission—see Table 8.

Control of Hemorrhage

Hemorrhage was controlled preoperatively in all 61 patients in the 5- to 10-pint group. Among the 77 patients in the 10- to 56-pint group, hemorrhage was not controlled prior to operation in 13, and control of hemorrhage preoperatively was questionable in 8. Of these 21, hemorrhage was brought under control at operation in all but four patients. Two of the four died on the operating table (one had a large laceration of the common iliac artery, the other had a high bilateral thigh amputation). One patient who sustained a massive liver wound died during the first postoperative day because of uncontrolled bleeding. The fourth patient had a massive liver and retroperitoneal muscle wound. He continued to bleed after operation, requiring 875 cc. of blood per hour to replace blood loss. He was re-explored 4 hours after the first operation. There were multiple bleeding sites in the retroperitoneal area, and this area was packed with two 5-yard-roll gauze packs. The oozing did not stop, however, until 7 pints of fresh blood had been administered. The patient subsequently recovered. This patient was given 46 pints of blood during the first 24-hour period.

In three other casualties who had uncontrolled hemorrhage preoperatively, the major points of hemorrhage were controlled at operation. Oozing continued from all surfaces, and they died after several hours. These casualties received 35, 52, and 56 pints of blood respectively.

Patients Admitted in Shock

In the group of 138 most severely wounded casualties, 33 were admitted in severe shock (Table 7); of this number, 7 died. Severe shock was a clinical diagnosis. Each of the 33 casualties had a systolic blood pressure of 80 mm. of mercury or less. The type of wound varied, but no particular type of injury predominated. The average time of evacuation was 2.3 hours. The total amount of blood

**Table 7. Most Severely Wounded—Admitted in Severe Shock
33 Patients—7 Deaths
(Case Fatality Rate—21 Per Cent)**

Patient	Type of Injury	Evac. Time (min.)	Admin. Blood Pressure	Preop. Blood (cc.)	Blood Total 1st 24 Hrs. (cc.)	Remarks
1	Extremity	110	70/30	2,000	2,500	Recovered
2	Extremity	105	80/40	2,000	3,000	Recovered
3	Abdominal	180	70/40	2,500	3,000	Recovered
4	Extremity	120	40/0	-----	3,500	Recovered
5	Abdominal	60	66/0	2,000	3,500	Recovered
6	Extremity	270	80/40	2,000	4,000	Recovered
7	Abdominal	185	60/30	2,500	4,000	Recovered
8	Extremity	270	70/40	3,750	4,750	Recovered
9	Chest	-----	60/0	2,500	3,500	Recovered
10	Abdominal	-----	40/0	3,000	5,000	Recovered
11	Thor-abd.	150	80/40	3,000	5,000	Recovered
12	Abdominal	-----	70/40	4,000	5,500	Recovered
13	Extremity	120	70/40	3,500	6,000	Recovered
14	Extremity	195	80/0	5,500	6,500	Recovered
15	Extremity	45	70/30	3,000	7,000	Recovered
16	Extremity	170	70/40	2,500	7,000	Recovered
17	Abdominal	130	70/0	4,000	9,000	Recovered
18	Abdominal	90	70/40	5,000	10,000	Recovered
19	Abdominal	90	74/52	3,500	11,500	Recovered
20	Extremity	103	0/0	5,500	5,500	Recovered
21	Abdominal	180	0/0	4,000	6,000	Recovered
22	Extremity	120	0/0	6,000	6,000	Recovered
23	Chest	190	0/0	4,000	7,000	Recovered
24	Abdominal	180	0/0	6,500	8,500	Recovered
25	Abdominal	-----	0/0	2,500	11,000	Recovered
26	Chest	70	0/0	5,500	13,000	Recovered
27	Thor-abd.	205	0/0	4,000	8,000	Expired, unknown
28	Abdominal	105	0/0	5,500	9,000	Expired, uncontrolled hemorrhage
29	Extremity	125	0/0	12,000	16,000	Expired, uncontrolled hemorrhage
30	Extremity	330	80/60	2,500	6,000	Expired, cardiac arrest
31	Extremity	90	80/60	5,500	9,500	Expired, postoperative shock
32	Extremity	85	40/0	5,500	11,500	Expired, undetermined
33	Abdominal	180	50/30	12,000	28,000	Expired, uncontrolled oozing
Average		140		4,300	7,600	

given in the first 24 hours ranged from 2,500 cc. to 28,000 cc. The average amount of blood administered preoperatively was 4,300 cc.; while the average for the first 24 hours was 7,600 cc.

Ten casualties in the shock group were admitted with blood pressure too low to be obtained by the usual cuff method. Three of these died, two from uncontrolled hemorrhage; the cause of death of the other casualty was unknown. The seven casualties who lived required from 11 to 26 pints of blood in the first 24 hours.

The causes of the other four deaths in this severely shocked group were cardiac arrest, refractory postoperative shock, uncontrollable oozing, and—in one instance—the cause of death could not be determined. In the 33 most severely wounded patients who were admitted in severe shock and required an average of 15 pints of blood, the case fatality rate was 21 per cent.

Blood Volume Data

Blood volume determinations were carried out in 15 casualties (Table 8).² The chromium 51-labeled, red-cell method was used because previous experience had suggested that it was more accurate than the T-1824 method (Evans blue dye). All determinations were performed in the early postoperative period. Because of continued hemorrhage, it is difficult to obtain accurate estimations of blood volume by any technic during the preoperative period. It was believed that postoperative volumes would give a good indication of blood loss. This seems to be a valid deduction based on the following statement. If a normal patient received 10 pints of blood during resuscitation and operation and his postoperative blood volume was approximately normal, it was assumed that blood loss was at least 10 pints.²

Table 8. Postoperative Cr 51 Blood Volumes ²

Patient	Type of Injury	Blood Received (pints)	Hct.	Postoperative Blood Volume (% of normal)*	Remarks
1	Abdominal	21	36.5	80	Recovered
2	Chest	20	28.0	48	Recovered
3	Extremity	19	32.0	78	Recovered
4	Chest	15	43.0	65	Cardiac arrest, recovered
5	Abdominal	15	43.0	82	Recovered
6	Extremity	14	40.0	62	Recovered
7	Thor-abd.	14	57.0	120	Recovered
7	9th post-op. day		41.0	107	
8	Abdominal	14	68.0	104	Expired 8th postop. day, peritonitis
9	Thor-abd.	12	48.5	% not calculated (b.v. 3,832 cc.)	Expired, postoperative shock
10	Abdominal	12	43.5	94	Recovered
10	(preop.)	1	42.5	57	
11	Abdominal	12	38.0	65	Recovered
12	Extremity	8	32.0	65	Recovered
13	Extremity	7	40.0	72	Recovered
14	Extremity	5	31.0	76	Recovered
15	Abdominal	5	35.0	96	Recovered

*Normal calculated on basis of 70 cc. per kilogram of body weight.

Determination of postoperative blood volume in these 15 casualties, after administration of massive quantities of blood, demonstrated an average blood volume of only 80 per cent of the calculated normal at the time each determination was carried out. Only one of these pa-

tients exhibited clinical evidence of shock. Patient 2 had a severe wound of the chest and of all extremities, resulting in massive bleeding into the chest and continued oozing from many areas in the subcutaneous tissues and muscles of the extremities. He received 20 pints of blood before and during operation. His condition postoperatively was unstable, and his blood volume was only 48 per cent of normal; hence an additional 6 pints of blood were administered. He progressed to an uneventful recovery.

Two casualties had blood volumes somewhat higher than normal. After receiving 14 pints of blood, Patient 7 had a hematocrit of 57 per cent and a blood volume of 120 per cent of the calculated normal. On the ninth postoperative day, his blood volume had decreased to 107 per cent of normal. Patient 8, suffering from an abdominal wound, received 14 pints of blood. At the end of operation, he had a hematocrit of 68 and a blood volume of 104 per cent of normal. He died later from an overwhelming peritonitis and mediastinitis.

It is interesting that, after administration of 1 pint of blood prior to operation, Patient 10 had a hematocrit of 42.5 and a blood volume of 57 per cent of normal. After resuscitation with 12 pints of blood, his hematocrit was 43.5; but his blood volume was only 94 per cent of normal.

Deaths

There were 20 deaths in the group of 138 most severely wounded casualties (Table 9). Most of this group were held at the hospital for a period of from 5 to 8 days and evacuated only after their condition became stable. Soon after injury, a few casualties were evacuated.

Table 9. Cause of Death

Etiology	Number of Deaths*
Uncontrolled oozing postoperatively	4
Postoperative shock	3
Uncontrolled hemorrhage	3
Peritonitis	2
Cardiac arrest	2
Massive lung damage	1
Pancreatitis	1
Septicemia	1
Aspiration pneumonia	1
Undetermined	1
Unknown	1
Total	20

* Short case histories of each patient who died appear at the end of this chapter.

uated to the Renal Insufficiency Center at the 11th Evacuation Hospital because of posttraumatic renal insufficiency. Deaths listed include those that occurred at the 46th Surgical Hospital and at the Renal Insufficiency Center. Renal failure was not listed as a cause of death in these patients because it was considered a contributing factor rather than a primary cause of death.

Causes of death were determined by clinical review of the case histories and available autopsy findings.

Eighty-nine Casualties Who Received Fifteen or More Pints of Blood or Plasma Expander

During 1952 and the first half of 1953, 89 battle casualties were studied during the first 24 hours after injury. Each of these patients required a minimum of 15 pints of blood or plasma expander—one and one-half times the normal blood volume. Because of the severity of the injury and the unusual amount of blood required for restoration, it was believed that a survey of the management and mortality of these casualties would yield valuable information. The patients who required 15 or more pints of blood or plasma expander from the previous group of 138 casualties were also included in this group of 89 casualties.

Some patients received 30, 40, and even 50 pints of resuscitative fluids within the first 24 hours. Of the patients who received more than 15 pints of blood or plasma expander, the case fatality rate was 44 per cent (Table 10). There was a wide difference in mortality

Table 10. Case Fatality Rate of Casualties Who Required 15 or More Pints of Blood and Plasma Expander (January 1952 to August 1953)

Type of Injury	Total			Number Dying of Continued Hemorrhage	Case Fatality Rate Excluding Continued Hemorrhage*
	No.	Deaths	Case Fatality Rates		
Abdominal	24	19	79%	11	61.5%
Abdominal and extremities.	29	12	41%	3	35.0%
Extremities	29	5	17%	1	14.0%
Chest	7	3	43%	1	33.0%
Total Average	89	39	44%	16	31.5%

*Case fatality rate computed after the number dying from continued hemorrhage had been excluded from the original total number of deaths.

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Table 11. Casualties Admitted with an Unobtainable Blood Pressure (January 1952 to August 1953)

Injury	Total		Receiving 15 or More Pints of Blood			Receiving Less Than 15 Pints of Blood		
	Number	Case Fatality Rate	Number	Deaths	Case Fatality Rate	Number	Deaths	Case Fatality Rate
Abdominal only	14	65.0%	10	9	90%	4	0	0
Abdominal and extremity	8	37.5%	7	3	43%	1	0	0
Extremity only	16	12.5%	9	2	22%	7	0	0
Average Total	38	37.0% -----	26	14	54% -----	12	0	0

between the patients who had injuries limited to the abdomen (79 per cent) and those who had injuries limited to the extremities (17 per cent). This difference cannot be explained entirely on the basis of uncontrollable, continued hemorrhage. When the patients were excluded in whom continued hemorrhage was the obvious cause of death, there remained a difference in case fatality rate between the abdominal and the extremity groups of 61.5 per cent and 14 per cent respectively.

The prognosis can be determined more accurately by the amount of blood required for resuscitation than by the degree of hypotension on admission to the hospital. This analysis is shown in Table 11. There were 38 casualties who had an unobtainable blood pressure on admission to the hospital. Of this number, 12 patients required less than 15 pints of blood during the first 24 hours; 26 patients required more than 15 pints. There were no deaths among the 12 patients who had an unobtainable blood pressure on admission and required less than 15 pints of blood. In contrast, the 26 casualties who required more than 15 pints of blood had a case fatality rate of 54 per cent.

Incidence of Renal Insufficiency

During 1952, 60 of the 89 casualties who required more than 15 pints of blood or plasma expander during the first 24 hours were studied consecutively in order to determine the incidence of renal insufficiency. Among these 60 patients, 43 lived for 3 days or longer. Of these 43, 21 per cent developed oliguria (output of urine amounting to less than 500 cc. per day) and 14 per cent developed nonoliguric azotemia (non-protein nitrogen of 80 mg. per cent or higher and output of urine amounting to more than 500 cc. per day). These statistics are given

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Table 12. Incidence of Renal Insufficiency in Battle Casualties Who Required 15 or More Pints of Blood and Lived Three Days or Longer*

Type of injury	Number Living 3 Days or Longer	Anuria (percent-age)	Oliguria (percent-age)	Nonoliguric Azotemia (percent-age)
Abdominal	9	22	11	11
Abdominal and extremity	14	28	0	7
Extremity	19	0	11	22
Chest	1	0	0	0
Total	43			
Average		14	7	14

*Based on 60 consecutive casualties requiring over 15 pints of blood. Thus, of the 43 casualties who lived, 35 per cent (anuria 14%, oliguria 7%, and azotemia 14%) developed clinically significant posttraumatic renal insufficiency.

in Table 12. However, 35 per cent of the 43 patients who lived 3 days or longer developed clinically significant post-traumatic renal insufficiency.

Discussion

In order to give perspective to this experience, a survey was made of 995 consecutive casualties admitted to the general surgical service between February and August 1953. Of these 995 patients, 138 (14 per cent) required replacement of 5 or more pints of blood. An analysis of the 138 patients demonstrated the major problems encountered in approximately 1,000 consecutive battle casualties treated. Among the 995 patients, 27 (3 per cent) required more than 15 pints of blood and plasma expander during the first 24 hours.

Mortality

Many of the casualties listed in Table 10 were so critically injured that they would not have reached the hospital alive had it not been for selective helicopter evacuation. Some of these casualties required only 15 minutes for actual transportation to the hospital. In the first 24 hours after injury, 89 patients received a total of 2,135 pints of type-O blood. This represents an average transfusion of 24 pints, or replacement of almost two and one-half times their blood volume. A case fatality rate of 44 per cent in this group is not surprising. The unusual result is that 56 per cent of these men with such massive wounds were transported to the hospital and were treated successfully.

Those patients having abdominal wounds had the highest case fatality rate (*see* Tables 10 and 11). Most of the deaths occurred during

the first 48 hours after injury. In the patients having wounds of the abdomen and requiring 15 or more pints of blood, the case fatality rate was 79 per cent; whereas, among casualties who had comparable wounds limited to the extremities, it was 17 per cent.

Hemorrhage from severe wounds of the extremity can be controlled by a tourniquet while replacement therapy is being carried out. As a result, anesthesia and operation may be undertaken in the presence of a relatively normal blood volume. In contradistinction, patients who have abdominal hemorrhage may not have the benefit of hemostasis prior to operation. In spite of pressure transfusions through 3 or 4 cannulae, the patient is subjected to the further trauma of anesthesia and operation in the presence of a marked deficit of blood volume. Emergency operation is mandatory on the patient who is such a grave risk, because it is impossible to correct the deficit until hemostasis can be achieved by operative means. The high mortality associated with abdominal injury represents, in part, the difficulty in controlling intra-abdominal hemorrhage. Obviously, several other factors contribute to this higher mortality, particularly the altered physiology associated with abdominal injuries.

Comparative Experiences

Discussing the experiences of the Second Auxiliary Surgical Group in World War II, Beebe and De Bakey point out that the case fatality rate was 21 per cent for all patients having abdominal injuries who were operated upon within 7 hours after injury. It is composed only of the 14 per cent of patients who represented the most severely wounded. The case fatality rate for those patients who had abdominal wounds was 16 per cent.³

Grant and Reeve reported on 59 battle casualties of World War II who had very large wounds of the extremities. The case fatality rate was 40 per cent.¹ In our survey of 52 battle casualties who had extremity wounds large enough to require replacement of 5 or more pints of blood, the case fatality rate was 10 per cent (*see* Table 2). In our group of 89 who required 15 pints or more of blood, there were 28 patients with wounds limited to the extremities (*see* Table 10). The case fatality rate was 17 percent in this group.

Undoubtedly this reduction in mortality has been influenced appreciably by the shorter evacuation time; but great credit must be given to the more vigorous use of whole blood.

Condition on Admission

Thirty-eight casualties were admitted to the hospital with a blood pressure so low that it was unobtainable by the usual cuff method. The results of therapy indicated a striking demonstration of the difference between severe hypotension on admission and severity of the

wound. There were no deaths among the 12 patients who received less than 15 pints of blood. Profound hypotension on admission increased mortality as evidenced by a case fatality rate of 44 per cent in the over-all group consisting of 89 patients (15 or more pints, *see* Table 10) and the increased case fatality rate of 54 per cent in 26 casualties (15 or more pints, *see* Table 11) who had an unobtainable blood pressure on admission.

A battle casualty who has a simple, rapid, but severe hemorrhage from an arterial defect can be resuscitated easily after hemorrhage is arrested. On the other hand, the casualty who has multiple, extensive wounds and hemorrhage from all areas is much more difficult to resuscitate. He will continue to lose blood from all the destroyed tissue until operative correction is carried out. He requires more blood and more extensive surgery than the patient whose hypotension on admission is caused by hemorrhage from a small arterial defect. Prognosis of a patient who is admitted with an obtainable blood pressure depends, to a large extent, upon the degree of trauma. Moreover, the influence of abdominal injury on mortality is reflected in casualties who were admitted with an unobtainable blood pressure. The case fatality rate was 65 per cent among those who had only an abdominal injury, whereas the case fatality rate was much lower (12.5 per cent) among those who had only extremity injuries (*see* Table 11).

Acute Post-traumatic Renal Insufficiency

Among 60 consecutive battle casualties requiring more than 15 pints of blood on whom a study was made, 14 casualties (35 per cent) developed clinically significant post-traumatic renal insufficiency (*see* Table 12). Among the 122 patients receiving 5 or more pints of blood and surviving 48 hours or longer, 6 developed renal insufficiency and 4 died (*see* Table 1). Among the patients who received 20 or more pints of blood and survived for 48 hours, 30 per cent developed renal insufficiency. Although several factors contribute to the development of renal insufficiency, it becomes apparent that this complication is encountered primarily in patients who suffer extensive tissue damage.

Deaths

From a review of the case histories, it is evident that most of the casualties who died had truly massive wounds. It is difficult to assess an exact cause of death in each instance. It appears that inability to maintain an effective blood volume was responsible for at least half of the deaths. Three patients died of major hemorrhage that could not be controlled; four died of hemorrhage as a result of multiple bleeding points from which diffuse, persistent oozing occurred after massive transfusion; and three died of postoperative shock in spite of large transfusions.

The forward surgeon encounters no problem that is more difficult than the management of a patient sustaining massive intra-abdominal hemorrhage. Hemorrhage can be controlled if the surgeon is able to diagnose continued, intra-abdominal hemorrhage and locate the bleeding point immediately upon opening the abdomen. Of the three patients who died of uncontrolled hemorrhage, two had a laceration of large vessels within the abdomen. In the over-all series, one patient had a large laceration of the iliac artery that was recognized early; hemorrhage was controlled immediately on opening the abdomen; the artery was repaired; and the patient recovered.

The four patients who died from uncontrolled, postoperative oozing from many areas may have had a deficiency in the clotting mechanism. This problem deserves further study. All four of these patients had intra-abdominal bleeding during the postoperative period. Two of them had a ligation of the inferior vena cava which may have contributed to the persistent oozing after operation. Uncontrolled oozing was a complication that followed the administration of 20 or more pints of blood. In occasional instances the administration of fresh, whole blood appeared to be beneficial. One patient experienced persistent oozing after the administration of 39 pints of stored blood. The oozing ceased after the infusion of 6 units of fresh, whole blood; and the patient survived.

The four patients who died of postoperative shock were all critically injured. They were hypotensive prior to operation; but all responded to rapid transfusion. Hypotension recurred during operation, or immediately thereafter, and persisted in spite of restorative efforts. In these patients, the exact mechanism of death is unknown. Although large quantities of blood were given, it might have been an inadequate amount. Many of the blood volume studies suggested that a deficit persisted in spite of massive transfusions.

One patient died of pancreatitis following injury to the pancreas. Better drainage might have prevented this death. Two patients died as a late result of cardiac arrest during operation.

Infection caused 3 of the 20 deaths. Two of the deaths were caused by peritonitis, and one by septicemia; both followed very massive injuries.

The group of casualties outlined in Table 12 required more than 15 pints of blood or plasma expander. Of these 60 patients, 43 lived 3 days or longer. Among the 17 deaths, the predominant causes were continued hemorrhage, 8; secondary hemorrhage, 1; postoperative, refractory hypotension, 4; and post-traumatic, renal insufficiency, 4. These results emphasize the need for further knowledge of the technical aspects of maintaining an adequate circulation during the first 24 hours after injury. Particular emphasis must be placed on control of intra-abdominal hemorrhage as one of the fields in which further investigation is necessary.

Massive Transfusions

The wound is a dynamic injury. It is not an injury of only the moment when the missile strikes the tissue; but it is a continuing insult involving continued loss of blood and fluids from the circulation. Resuscitation, therefore, must be a continuing process, as was pointed out by Beecher.⁴ A person should not speak of resuscitation *and* surgery, as surgery is a vital part of resuscitation.

It was not surprising to note the large amounts of blood that were necessary during various phases of resuscitation of those patients who required massive transfusions. In this group, the amount of blood given preoperatively for correction of deficit in blood volume was equal to that given during surgery. During the postoperative period, a number of patients required about one-third of the amount of blood given during operation.

Emphasis must be placed on the large amount of blood required prior to operation. In the most seriously injured group (10 to 35 pints), an average of 7 pints was given before the patient was taken to surgery. During operation, 6 pints were administered; and during the immediate postoperative period 38 of the patients required an additional 2 pints of blood. A similar ratio was found in the 5- to 10-pint group, that is 3.5 pints preoperatively and 3 pints during operation. Eighteen of the casualties required an average of 1.5 pints during the immediate postoperative period (*see* Table 5). The blood volume of a patient on admission to a medical installation is not an indication of the amount of blood that will be required for total resuscitation. Half of the blood volume may have been lost before a patient arrives at a hospital; but there is also a continued loss during the preoperative period and during surgery. Absolute control of hemorrhage in the severely injured battle casualty is most difficult to achieve.

The amount and rate of administration of blood during operation may be determined by the blood loss that occurs during the procedure; and underestimation of this loss is common. Restoration of blood volume does not terminate at the end of the surgical procedure. It would be ideal *if* a patient could be restored to a normal blood volume before surgery and *if* normal blood volume could be maintained by further transfusions as blood is lost during the operation. Unfortunately, this situation is most difficult to achieve in the critically injured soldier. Determination of the amount of blood required is based on: (1) response of the patient (blood pressure, pulse rate, output of urine, and response to anesthesia and to changes in posture) and (2) clinical judgment of the surgeon (observed blood loss, color of conjunctiva, and extent of tissue damage). In many instances, additional blood is necessary postoperatively either because restora-

tion has not been complete or because the patient continues to lose blood and plasma during the postoperative period. A persistently low blood pressure nearly always indicates the need for further transfusion.

The successful use of massive transfusions re-emphasizes certain lessons learned in World War II, namely, the treatment of wound shock is primarily the control of hemorrhage and the administration of adequate quantities of blood. One of the primary differences between resuscitation during the Korean conflict and during World War II was a difference in the amount of blood administered. Massive transfusions were used frequently and only rarely was there evidence of overtransfusion. From the data on blood-volume determinations after massive transfusions, it appears that large quantities of blood were lost from the effective circulation. In most instances, recovery from initial hypotension followed immediately after the administration of large quantities of blood. An occasional patient who reached the hospital alive, but who had vital organ damage, died even though there was only a minimal blood loss. This was observed most frequently in battle casualties who sustained injuries of the brain. In general, the severity of the injury was directly proportionate to the amount of blood lost. Both the injury and blood loss continued to exert a deleterious effect.

The impressions gained from the observations in this report parallel those of the Board for the Study of the Severely Wounded: "If irreversible shock was present prior to anesthesia, we missed it. If toxins caused any of the shock immediately after the injury, we failed to recognize it."⁴

A diagnosis of irreversible shock should not be made prior to death. Under the conditions of this study, continued hypotension prior to anesthesia and in the absence of injury to the central nervous system or heart was due to continued hemorrhage or inadequate transfusion.

Summary and Conclusions

A survey of experiences with the most severely wounded patients undergoing general surgical treatment was conducted at the 46th Surgical Hospital in Korea. The amount of blood required for resuscitation was used to categorize extent of injury. The case fatality rate for the battle casualties who required 15 or more pints of blood was 44 per cent. The average time of evacuation was 3.8 hours.

In all categories, mortality was higher for casualties who had wounds of the abdomen than for those who had wounds of the extremities.

Because of continuing hemorrhage, the amount of blood required during operation was equal to the amount required preoperatively;

and the amount required postoperatively was directly proportionate to the extent of tissue damage.

It was found desirable to restore a blood volume prior to operation but, in some instances and particularly in abdominal injuries, an emergency operation was necessary for the control of hemorrhage.

Thirty-three patients were admitted with a systolic blood pressure of 90 mm. of mercury or less and the amount of blood required averaged 15 pints. Case fatality rate for this group was 21 per cent. The prognosis could be better predicated by the amount of blood required than by the degree of hypotension on admission.

Blood volume determinations by the labeled, red-cell method showed that overtransfusion rarely occurred.

Twenty-one per cent of the casualties who required 15 or more pints of blood developed severe oliguria (output of urine amounting to less than 500 cc. per day).

Inability to maintain an effective blood volume (uncontrolled oozing, uncontrolled hemorrhage, postoperative shock) was the cause of half of the deaths.

Case Reports

Case 1. (Peritonitis, transfused 9 pints of blood)

This 23-year-old Korean soldier was injured by mortar-shell fragments on June 27 at 0950 hours. He was evacuated to a clearing station and then by helicopter to the 46th Surgical Hospital 3.5 hours after injury. The injuries involved his back and abdomen. On admission to the hospital, his blood pressure was 120/76, pulse 84; while at the battalion aid station his blood pressure was 70/40. Preoperatively 1,500 cc. of blood were administered; then the patient was taken to surgery 1.5 hours after admission to the hospital.

Laparotomy showed laceration of the duodenum, cecum, and the left kidney. The duodenal lacerations were sutured; the posterior cecal perforation was closed; and a tube cecostomy was performed through the anterior perforation. The right perirenal area was drained posteriorly. During the 3-hour operation, 5 pints of blood were given. Blood pressure fell to 82/40 for approximately 10 minutes during surgery. Because of unstable blood pressure, it was decided not to turn the patient over in order to débride his back wounds. He developed oliguria postoperatively, passing 139 cc. of urine on the first postoperative day and 57 cc. on the second postoperative day.

Because of renal difficulties, the patient was transferred to the Renal Insufficiency Center on June 29. He was placed on a regimen of 25 percent glucose with insulin and vitamins. There was no laboratory evidence of hyperkalemia. He developed Cheyne-Stokes breathing during the night; his blood pressure fell, and signs of pulmonary edema developed. On June 29, 1953, he suddenly expired. The staff felt that death was caused by peritonitis and that renal insufficiency played only a minor role in the cause of death.

Case 2. (Cardiac arrest, transfused 10 pints of blood)

This 39-year-old Korean soldier was injured by mortar-shell fragments on May 21, at 1200 hours. He was evacuated to a clearing station, and then brought by ambulance to the 46th Surgical Hospital 4 hours after injury. He sus-

tained penetrating wounds of the right chest, abdomen, open, comminuted fractures of the right humerus. At the time of admission, his blood pressure was 98/70, pulse 112. He was given 1,500 cc. of blood preoperatively and then taken to surgery 1.3 hours after admission to the hospital (5.4 hours following injury). During the surgical procedure there was cardiac arrest and cardiac massage was performed to re-establish cardiac rhythm. The chest wound was débrided and a thoracotomy tube was inserted. An exploratory laparotomy revealed a laceration of the liver; this area was drained with Penrose drains. During the 4-hour operation, 2,500 cc. of blood were given.

Bleeding through endotracheal tube occurred postoperatively. An electrocardiogram taken on the first postoperative day was essentially normal. The patient expired suddenly 19 hours after completion of the operation. It was believed that death resulted from a second cardiac arrest.

Case 3. (Shock, transfused 10 pints of blood)

This 26-year-old Korean soldier was injured by mortar-shell fragments on June 20 at 1000 hours. He was evacuated by ambulance from a battalion aid station to the 46th Surgical Hospital, arriving 8.75 hours after injury. He sustained penetrating wounds of the left arm, leg and face, and partial traumatic amputation of the left hand. On admission, his blood pressure was 80/?. He was given 2,500 cc. of blood preoperatively and taken to surgery 1.25 hours after admission to the hospital.

This patient's face, neck, leg, and foot were débrided. The left posterior tibial artery was ligated and the left hand was amputated. During the 1.5-hour operation, 1,500 cc. of blood were administered. His blood pressure remained around 80/60 part of the time during surgical intervention and, for approximately 36 minutes, it was unobtainable. On completion of the operative procedure, his blood pressure was 104/62, pulse 100.

The patient's postoperative course was unsatisfactory. Twenty-four hours following surgery, his blood pressure fell to 60/40 and norepinephrine was given for the next 16 hours. Oliguria was noted for a 12-hour period during this 16-hour interval. Twenty-four hours after completion of the operation, his temperature was 104° F.; and, after 36 hours, it was 102° F. He died in shock a few hours later.

Case 4. (Cardiac arrest, transfused 12 pints of blood)

This 22-year-old American soldier was injured by mortar-shell fragments on July 20 at 2300 hours. He was brought by ambulance from a collecting station to the 46th Surgical Hospital at 0230 hours, 3.5 hours after injury. On admission, his blood pressure was 0/0. He had penetrating wounds of the neck, face and left arm, and perforating wounds of both legs. Prior to admission, he had received 500 cc. of blood, 500 cc. of dextran, and 100 cc. of albumin. He was given 2,000 cc. of blood prior to surgery.

This patient had cardiac standstill after induction of anesthesia, and cardiac massage was begun to restore cardiac rhythm. Débridement and an above-the-knee guillotine amputation of the right leg was performed. He was given 2,000 cc. of blood during the operation. Blood pressure was below 80 systolic briefly during this time and for the entire 12-hour postoperative period, at which time the patient expired. He remained comatose after surgery; and there was progressive oliguria. Temperature at the time of death was 106.8° F. The patient did not appear to recover from the systemic effects of cardiac arrest.

From injury to the hour of his death, this patient was given a total of 6,000 cc. of blood.

Case 5. (Shock, transfused 12 pints of blood)

This 25-year-old Korean soldier was injured by mortar-shell fragments on June 4 at 0800 hours. He was evacuated from a battalion aid station to the 46th Surgical Hospital where he arrived at 1055 hours, 2.9 hours after injury. On admission to the hospital, his blood pressure was 90/40, pulse 120. Blood transfusion was started, but the blood pressure fell to 60. After 2,500 cc. of blood and 1,000 cc. of dextran had been given, his blood pressure rose to 120/70; and he was taken to the operating room.

Abdominal exploration revealed large amounts of a dark liquid, resembling bile, in the peritoneal cavity. Active bleeding was present in the retroperitoneal area. There were perforations of the diaphragm, of the right lobe of the liver with necrosis of liver substance, and of hepatic flexure of the colon, tangential tear of the duodenum, avulsion of two lumbar veins, a small laceration in a vein near the adrenal gland, and a perforation of terminal ileum in two places. All tears and perforations were sutured; bleeding vessels were ligated; the perforated colon was exteriorized; and chest wounds were débrided. During the 2-hour operation, 2,500 cc. of blood were given.

Postoperatively the patient was hypotensive for 20 hours; and his blood pressure varied from 70 to 86 systolic. Norepinephrine was started and continued at a slow drip to maintain the blood pressure around 100 systolic. For the first 8 hours, output of urine was 120 cc., and it was 90 cc. for the next 12 hours. A tracheotomy was performed because of respiratory distress. The patient's progress was very unsatisfactory. Elevation of serum potassium continued and oliguria persisted. The staff decided to evacuate this patient to the Renal Insufficiency Center, but he died en route.

Case 6. (Aspiration pneumonia, transfused 12 pints of blood)

This 23-year-old American soldier was injured by mortar-shell fragments on June 11 at 0530 hours. He was taken to the 44th Surgical Hospital; and his initial blood pressure was 76/40. Injuries consisted of traumatic amputation of the right forearm, partial penetrating wound of the abdomen, open comminuted fracture of the right humerus, fracture of the right tibia, and multiple wounds of the arms and legs. He was given 3,500 cc. of blood. It was decided to transfer this patient by helicopter to the 46th Surgical Hospital. He was given another 500 cc. of blood and taken to the operating room. At surgery, his blood pressure was 118/80.

An exploratory laparotomy revealed a large perforation of the sigmoid colon; this was exteriorized. No other organ damage was found. During the closure of the abdomen, the patient's general condition deteriorated. On several occasions during surgery his blood pressure fell to 84/60. Débridement of wounds of the right forearm was carried out rapidly. Because of the patient's critical condition, it was felt that a complete débridement should not be performed. During the 2.5-hour operation, 2,000 cc. of blood were given.

While recovering from the anesthesia, the patient vomited and apparently aspirated some of the vomitus. Bilateral bronchopneumonia rapidly developed. Postoperatively the patient remained comatose; and he died on the fifth post-operative day.

Case 7. (Massive lung damage, transfused 13 pints of blood)

This 20-year-old American soldier was wounded by enemy gun fire on May 6 at 0500 hours. He was given 500 cc. of dextran and 400 cc. of blood at a battalion aid station. Upon arrival at the collecting station, his blood pressure was 106/70, and he was given another 500 cc. of dextran. He was brought by ambulance of the 46th Surgical Hospital at 0840 hours, 3.7 hours after injury.

He had sustained a penetrating wound of the left chest. On admission, his blood pressure was 174/90, pulse 140. Respiration was rapid and labored; but there was no cyanosis. The patient was taken to the operating room and, when he was turned on his side following induction of anesthesia, there was a fall in blood pressure. He was given a total of 13 pints of blood in an attempt to treat the hypotension. Pulmonary edema developed within a short time, and the patient died before the operation was actually begun. It was felt that he died because of extensive damage to a vital organ.

Case 8. (Mediastinitis, transfused 13 pints of blood)

This 22-year-old American soldier was injured by mortar-shell fragments on July 5 at 0200 hours. He was evacuated by helicopter to the 46th Surgical Hospital 4.75 hours after injury. He had a penetrating wound of the abdomen. On admission, his blood pressure was 100/80, pulse 120. Preoperatively he was given 3,250 cc. of blood; and he was taken to surgery 4.5 hours after admission to the hospital.

Laparotomy revealed a laceration of the cecum and multiple lacerations of the small bowel. Two resections of the small bowel were made and a cecostomy was performed. During the 3-hour operation, he was given 3,500 cc. of blood. His blood pressure was unobtainable on two occasions during surgery. Norepinephrine was begun as soon as he was returned from the operating room.

Postoperatively the patient did very poorly. A diagnosis was made of lower left lobar pneumonia. There was persistent hypotension and oliguria. The patient also developed signs of a pulmonary infarct. Clinical jaundice developed on the fifth day after operation. There was an episode of hematemesis on the seventh postoperative day. The patient was moved to the Renal Insufficiency Center where further resuscitative measures were carried out with blood. He continued to vomit dark-colored matter. A Levine tube was passed into the stomach and, when an x-ray was taken, the tube was shown to be coiled in the right mediastinum. Apparently there was an unrecognized perforation of the esophagus. The patient's condition deteriorated and he died on July 13, the eighth day after injury.

Case 9. (Pancreatitis, transfused 14 pints of blood)

This 21-year-old American soldier was injured by small arms fire on May 12 at 2016 hours. He was taken to a battalion aid station and then evacuated to the 46th Surgical Hospital, arriving 2.25 hours after injury. He had a perforating gunshot wound of the upper abdomen. He was given 500 cc. of dextran at a battalion aid station. On admission to the hospital, his blood pressure was 120/60, pulse 84. Preoperatively he was given 3 bottles of blood (1,500 cc.) and taken to the operating room 2.58 hours after admission.

Laparotomy revealed perforations of the stomach, splenic flexure of the colon, and lacerations of the spleen and left kidney. There was a slight injury to the tail of the pancreas. The stomach perforations were closed; the spleen and left kidney removed; the splenic flexure of the colon was resected; and a double-barrel colostomy performed. On several occasions during the operation the blood pressure fell; but it stabilized following administration of 9 bottles of blood and norepinephrine intravenously. The abdomen was drained with one Penrose drain in the left gutter through the wound of exit in the left lateral lumbar area.

The patient progressed satisfactorily until the evening of the 18th of May, the sixth day after injury, when he became distended and somewhat irrational. The following day he became anuric, hypotensive, and had a convulsive seizure. It was felt that the patient had pancreatitis along with renal insufficiency. He

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continued to have tetanic muscular contractions and he died at 1400 hours on May 19th.

Postmortem examination showed an inflammatory pancreatitis with necrosis. The right kidney was large, soft, and edematous.

Case 10. (Shock, transfused 16 pints of blood)

This 41-year-old Korean soldier was injured by mortar-shell fragments on April 22 at 0545 hours. He sustained penetrating wounds of the chest and abdomen, perforating wounds of the right arm. He received 1,000 cc. of dextran at a battalion aid station and 500 cc. of blood at a collecting station. He was then evacuated to the field unit of the 46th Surgical Hospital, arriving 2.4 hours after injury. A sucking wound of the chest had been closed with gauze. On admission, his blood pressure was 0/0, pulse 98. He was pale and cold; and he had labored respiration and a barely perceptible pulse. He was given 8 pints of blood intravenously and intra-arterially before being taken to surgery 5 hours after admission to the field unit. Lung tears were found at operation; and these were sutured. After the lungs were re-expanded, the chest was closed. Laparotomy revealed large amounts of free blood, a perforating wound of the hepatic flexure of the colon, a perforation of the duodenum, and retroperitoneal hemorrhage. The hepatic flexure was exteriorized and the other perforation was closed. During the operation, blood pressure varied between 116/70 and 78/58; and 8 pints of blood were transfused.

Postoperatively the patient's blood pressure ranged from 100/60 to 84/50; his respiration was 48, and output of urine on the day of surgery totaled 350 cc. On the first postoperative day he was moved from the field unit of the 46th Surgical Hospital to the rear unit. His blood pressure at this time was ranging from 90/60 to 80/50. He died a few hours later in shock.

Case 11. (Shock, transfused 19 pints of blood)

This 21-year-old American soldier was struck by mortar-shell fragments on June 19 at 0655 hours. He sustained penetrating wounds of the face and hands, open comminuted fractures of the right and left tibia, traumatic amputation of the right hand, and multiple superficial injuries. The soft tissue of both legs was damaged extensively. This patient was evacuated by helicopter to the 46th Surgical Hospital, arriving 1.5 hours after injury. At a battalion aid station this patient had received 1,000 cc. of dextran. On arrival at the surgical hospital, his blood pressure was 80/60, pulse 90. Preoperatively he received 5,500 cc. of blood. He was then taken to surgery 6.5 hours after admission to the hospital.

A below-knee amputation was performed on the left leg; and the right hand was amputated. Extensive débridement was performed on wounds of the left leg, and a cast was applied. Remaining multiple wounds were also débrided. For 4 hours prior to surgery, the patient's blood pressure was below 80; but it remained around 110 during the 1.5-hour operation. Pulse varied from 144 to 160.

Postoperatively the patient's blood pressure was below 80 most of the time, even though norepinephrine was given. There was marked hemoglobinuria; and the patient died 20 hours following completion of surgery.

Case 12. (Uncontrolled hemorrhage, transfused 19 pints of blood)

This 24-year-old American soldier was injured by mortar-shell fragments on May 26 at 0845 hours. He sustained penetrating wounds of the neck, abdomen, both arms and legs. He was taken to a battalion aid station where 500 cc. of dextran were administered; and then evacuated by helicopter to the 46th

Surgical Hospital, arriving 2 hours after injury. Eleven pints of blood were given prior to the operation.

An abdominal exploration revealed a large amount of blood in the peritoneal cavity and a huge retroperitoneal hematoma. There was a laceration of the common iliac artery and a superior mesenteric artery thrombosis. When the large retroperitoneal hematoma was evacuated, the patient suddenly died. During the 50-minute surgical procedure, 8 pints of blood were given.

Case 13. (Septicemia, transfused 20 pints of blood)

This 22-year-old American soldier was injured by an M-1 rifle missile on February 24 at 0100 hours. The missile perforated the upper abdomen. He was taken to a battalion aid station; his blood pressure at that time was 115/65. At a collective station, this patient's blood pressure was 90/60, pulse 100. After receiving 500 cc. of blood, he was evacuated by ambulance to the 46th Surgical Hospital and arrived at the hospital 4.25 hours after injury. His blood pressure upon admission was 99/80. Preoperatively he was given 2,000 cc. of blood; then he was taken to the operating room 4.5 hours after admission to the hospital.

Laparotomy revealed four perforations of the hepatic flexure of the colon, and perforations of the right lobe of the liver and inferior pole of the right kidney. The colon was exteriorized; liver injuries were packed with gelfoam, and a small wedge was removed from the lower pole of the kidney. The peritoneal cavity was drained. Fifteen pints of blood were administered during surgery; but blood pressure remained around 80/50 throughout the procedure. Norepinephrine was also given.

Postoperatively the patient had a stormy course. Most of the time his blood pressure was over 100, but he became oliguric the first postoperative day. On the second postoperative day he was moved to the Renal Insufficiency Center. He was dialyzed on the artificial kidney once, but his condition continued to deteriorate. A blood culture showed that septicemia was present and the patient died on the 12th postoperative day.

Postmortem examination showed an abscess in the right kidney, peritonitis, and a large retroperitoneal hematoma.

Case 14. (Uncontrolled hemorrhage, transfused 22 pints of blood)

This 23-year-old American soldier was injured by mortar-shell fragments on May 7 at 0140 hours. Fragments penetrated the right arm, back, and right side. He was taken to a battalion aid station, and then to the clearing station where he was given 1,000 cc. of dextran. He was evacuated by ambulance to the 46th Surgical Hospital and admitted 4.7 hours after injury. On admission his blood pressure was a questionable 100, pulse 128. The patient had a sucking wound of the chest and gross hematuria. He was given 3,500 cc. of blood prior to surgery.

The chest wound was débrided and closed. Laparotomy revealed lacerations of the diaphragm and liver, transection of the small bowel in three places, a perforation of the sigmoid colon, and a shattered left kidney. The tear in the diaphragm was closed, and the small bowel repaired; a left nephrectomy was performed; and a loop colostomy was made. During the 3-hour operation 3,000 cc. of blood were given; and for a period of 1.75 hours there was hypotension of less than 80.

Postoperatively the patient continued to bleed. During the first 16 hours following operation he was given 4,500 cc. of blood; and his output of urine was nil. Sixteen hours after completion of surgery this patient died. Apparently hemorrhage continued from the liver wound.

Case 15. (Undetermined, transfused 23 pints of blood)

This 21-year-old American soldier was injured accidentally by a grenade on July 28 at 1045 hours. Fragments struck his left hand, both eyes, buttocks, thighs, and legs. He was taken to a battalion aid station where 500 cc. of blood and 1,000 cc. of dextran were administered. Then he was evacuated by helicopter to the 46th Surgical Hospital, arriving 1.4 hours after injury. His blood pressure was 40/0 on admission. Preoperatively he was given 500 cc. of blood and taken to surgery 3.5 hours later.

At surgery an exploratory laparotomy was negative. Multiple débridements were carried out. The popliteal artery on the right side was injured and, in addition to extensive damage of the soft tissue, he had sustained fractures of the left fibula and of both the right and left tibia. The injured popliteal artery was anastomosed. During the 4-hour operation 5,000 cc. of blood were given.

Postoperatively the patient was quite ill. For 8 hours immediately following the operation his blood pressure was around 80 to 90. His right leg was cold below the knee; and output of urine was only 200 cc. the first postoperative day. Because of the patient's progressive deterioration, he was taken back to the operating room the second postoperative day and an above-the-knee amputation was performed on the right side. At this time his serum potassium was 8.6 milliequivalents per liter. Because of oliguria he was placed on a fluid restriction regimen. He was then evacuated to the Renal Insufficiency Center where the left leg was amputated because of extensive damage to tissue, as it was felt that massive tissue necrosis was contributing to the oliguria and hyperpotassemia. The following day the patient died.

Case 16. (Uncontrolled hemorrhage, transfused 32 pints of blood)

This Korean was struck by artillery-shell fragments on April 26 at 1040 hours and sustained traumatic amputation of both legs, penetrating wounds of both arms, and multiple superficial wounds. He was taken to a battalion aid station and then evacuated by helicopter to the 46th Surgical Hospital where he was admitted 2.08 hours after injury. On admission his blood pressure was 0/0. There was no perceptible pulse, and no reaction to painful stimuli. Blood was transfused intravenously and intra-arterially; and after 5 pints of blood had been given his blood pressure was 60/?. During a 5.3-hour resuscitative period he received 24 pints of blood; and when his blood pressure rose to 110/85 he was taken to surgery.

Six cc. of pentothal was all the anesthesia required. Four teams of surgeons reamputated both thighs and débrided both arms and hands in a total operative period of 30 minutes. After 21 pints of blood were transfused by drip, blood pressure fell to 70/0. By rapidly pumping in 1,000 cc. of blood, his pressure rose to 100/0; but he died on the operating table 30 minutes after surgery was completed.

Case 17. (Uncontrollable oozing of blood, transfused 33 pints of blood)

This 24-year-old American was injured by small arms fire on April 9 at 0300 hours. The missile penetrated the abdomen and back. Blood pressure at the battalion aid station was 110/74, pulse 84; and at the collecting station blood pressure was 40/0. The patient was given 1 bottle of blood (500 cc.) and evacuated to the clearing station where 2 more bottles of blood were given prior to evacuation to the 46th Surgical Hospital. On admission 4 hours after injury, blood pressure was 120/80, pulse 118. Within the next 2 hours he was given 2 more pints of blood and then taken to the operating room.

An exploratory laparotomy revealed perforations of the inferior vena cava, hilum of the left kidney, and of the duodenum. The vena cava was ligated

above the renal veins; the left kidney was removed; and the duodenum repaired. Blood pressure varied during surgery from 110 to 60/0. He was given 28 pints of blood during the operation and when the laparotomy wound was being closed, blood was oozing from multiple areas. He died approximately 5 hours after completion of the operation. He had been given a total of 33 pints of blood.

Case 18. (Uncontrolled oozing of blood, transfused 40 pints of blood)

This 20-year-old American soldier was struck in the abdomen by shell fragments on July 7 at 0415 hours. He was given 6 pints of blood at the collecting station prior to evacuation to the 46th Surgical Hospital. Upon admission to the hospital 5.75 hours following injury, his blood pressure was 100/64, pulse 64. One hour and 10 minutes after admission he was rushed to surgery.

Laparotomy revealed perforations of the interior vena cava, the right ureter, duodenum, and colon. The vena cava was ligated; ureter repaired over a catheter; and a nephrostomy performed. The colon was exteriorized. During operation 17 pints of blood were given; but the patient's blood pressure varied from 100 to 80/60.

Postoperatively he became oliguric. In the immediate 24 hours following surgery, the serum potassium was 9.2 milliequivalents per liter, and the electrocardiogram showed that myocardial changes had occurred. He was evacuated to the Renal Insufficiency Center where he was dialyzed on the artificial kidney on the second postoperative day. Serum potassium fell from 8 to 5.4 milliequivalents per liter. After dialysis blood pressure was very unstable and, although large amounts of blood were given, he continued to deteriorate and died on the morning of the third postoperative day.

Postmortem examination revealed a large amount of blood in the peritoneal cavity and evidences of bleeding from the psoas muscle and surrounding areas.

Case 19. (Uncontrollable oozing of blood, transfused 52 pints of blood)

This 36-year-old Korean was injured by mortar-shell fragments on June 26 at 2210 hours. He sustained penetrating wounds of the buttocks, pelvis, and a traumatic amputation of the right foot. Prior to admission to the hospital he had received 1,500 cc. of dextran. Four hours after injury he was brought by ambulance to the 46th Surgical Hospital. On admission his blood pressure was 110/60, pulse 110. Prior to operation, however, blood pressure fell; and during a resuscitative period of 4.5 hours before he was taken to surgery, he was given a total of 11 bottles of blood.

Examination revealed multiple perforations of the rectum. A diverting colostomy was made. All wounds were débrided. There was massive destruction of the gluteal muscles, fractures of the sacrum and right femur. A right, lower leg amputation was performed. During a 2.5-hour operation he was given 20 pints of blood.

Postoperatively there was hypotension, although more blood and norepinephrine were given. He died 11 hours following operation. A total of 52 pints of blood had been given. There was continued oozing from all wounds until death.

Case 20. (Uncontrollable oozing of blood, transfused 56 pints of blood)

This 21-year-old American soldier was injured by mortar-shell fragments on July 24 at 1730 hours. He sustained extensive multiple wounds of the lips, right arm, both thighs, and right buttock. He was evacuated by helicopter from the battalion aid station to the 46th Surgical Hospital, and arrived

3 hours after injury. On admission his blood pressure was 50/30. Preoperatively he was given 22 pints of blood and then taken to the operating room.

At surgery there was a laceration of the liver and two intraperitoneal rectal perforations. The rectal perforations were repaired, a diverting colostomy was performed and the liver was sutured. Because of the poor condition of the patient, the wounds of the lips, right arm, and thighs were not débrided. Six pints of blood were given during the operation.

Postoperatively for 9 hours there was hypotension below 80, and multiple transfusions were given. The patient failed to rally and continued to ooze from various wounds. He had received a total of 56 pints of blood from time of injury until he died 9 hours after operation.

Postmortem examination showed over 7 liters of blood in the peritoneal and pleural spaces. In addition, there had been bleeding into the retroperitoneal spaces and muscles.

References

1. Grant, R. T., and Reeve, E. B.: *Observations on the General Effects of Injury in Man: With Special Reference to Wound Shock*. Medical Research Council, Special Report No. 277, London, England.
2. Prentice, T. C.; Olney, J. M., Jr.; Artz, C. P., and Howard, J. M.: *Studies of Blood Volume and Transfusion Therapy in the Korean Battle Casualty*. Surg., Gynec. & Obst. 99: 542, 1954.
3. Beebe, G. W., and De Bakey, M. E.: *Battle Casualties*. Charles C Thomas, Springfield, Illinois, 1952.
4. Board for the Study of the Severely Wounded: *The Physiologic Effects of Wounds*. The Office of The Surgeon General, Department of the Army, Washington, D. C., 1952.

Chapter 3

Use of an Intra-Aortic Balloon Catheter Tamponade for Controlling Intra-Abdominal Hemorrhage in Man*

Lieutenant Colonel Carl W. Hughes, MC, USA

Everyone is familiar with the practice of manually compressing the exposed aorta for a period of time to control hemorrhage or to combat a hypotensive state during laparotomy. To accomplish this same result in the unexposed aorta, a balloon catheter was tested as an intra-aortic tamponade in two critically injured Korean casualties.

This device (Fig. 1) is a number 10, French, non-radio-opaque, Dotter-Lukas¹ balloon catheter, 125 cc. long, with a single lumen communicating with the balloon and equipped with Luer-Lok at the proximal end. The terminal end is occluded and rounded. The balloon begins 1 cm. from the end and is inflated by filling with 20 cc. of sterile normal saline (Fig. 2). It is sterilized by immersion in zephiran solution for 20 minutes.

The catheter was chosen for testing as an intra-aortic tamponade, primarily in those casualties with intra-abdominal hemorrhage whose shock could not be controlled by blood replacement. It was arbitrarily decided that the catheter would be used only in two categories: (1) in moribund patients with evidence of intra-abdominal bleeding in which blood pressure could not be obtained after administration of 10 units of blood; and (2) in moribund patients who were in shock from causes other than blood loss. The assumption was that temporary intra-aortic tamponade would enhance perfusion of the coronary arteries.²

Case Reports

Only three patients fitting the first category were observed.

CASE 1. The first patient in this category had a thoraco-abdominal wound. He received approximately 20 units of blood, with progressively increasing distention of the abdomen. The medical officer who was attempting to resuscitate the patient did not think to ask for use of the catheter. Instead, the patient was taken to surgery with an almost unobtainable blood pressure. The bleeding could not be controlled and the patient died on the table. Postmortem examination revealed a laceration of the splenic artery. This case represents the ideal type of case in which use of the catheter might have been lifesaving.

*Previously published in *Surgery* 36: 65, 1954.

Intra-aortic Balloon Catheter Tamponade

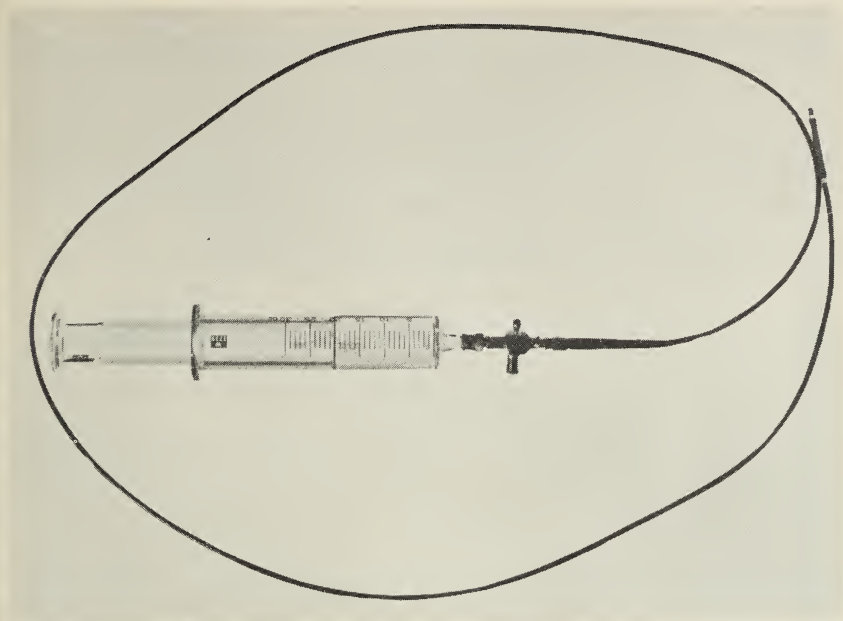


FIGURE 1. The catheter with balloon deflated.

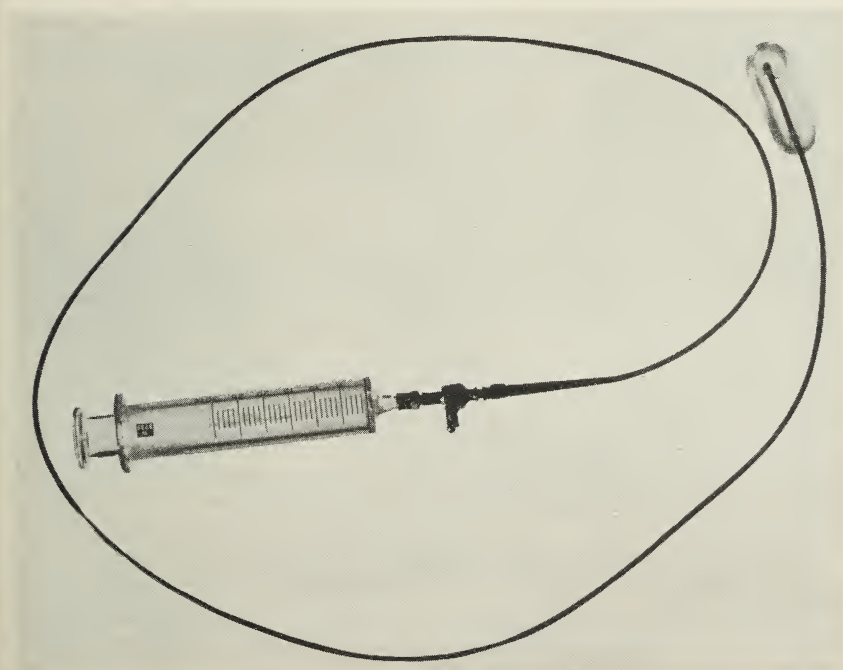


FIGURE 2. The catheter with balloon inflated with 20 cc. of sterile normal saline solution.

CASE 2. The second patient was admitted 1 hour after injury suffering from grenade wounds of the abdomen, thighs, leg, and foot, and an open comminuted fracture of the right tibia and fibula. He had received 1,000 cc. of plasma prior to admission. His blood pressure on admission was 40 systolic, with a questionable diastolic level; and pulse rate was 120. There was abdominal distention, suggesting uncontrolled intra-abdominal bleeding. In the receiving ward he was given 2,000 cc. of whole blood and 500 cc. of dextran; and his blood pressure was read as 58 systolic, with a questionable diastolic pressure, and pulse was 120. He received 2,500 cc. more of blood without improvement. He was then taken to surgery in a moribund condition; and blood pressure could not be obtained. The level of the diaphragm was estimated by measuring the catheter beside the patient from the common femoral artery to the ninth-rib level in the mid-axillary line. Two umbilical tapes were placed about the common femoral artery and a small transverse slit was made in the artery between the tapes. Through this slit, the catheter was inserted and passed to the level of the diaphragm where the balloon was inflated with 20 cc. of normal saline. His blood pressure was read immediately as 110/70, and his respiration improved. With the catheter in place, the abdomen was opened quickly and from 1,500 to 2,000 cc. of free blood was aspirated. A bleeding, external iliac vein was controlled and a massive laceration of the right lobe of the liver was packed and sutured.

After 15 minutes, the catheter was slowly deflated; then it was moved down to the bifurcation of the aorta and reinflated to allow oxygenation of the kidneys, liver, and spinal cord. This procedure allowed bleeding at multiple points in the bowel and mesentery and around the celiac axis. Even after bleeding, the patient's blood pressure was not obtainable and breathing became labored. The catheter was then replaced and inflated at the level of the diaphragm. A blood pressure of 78/50 was obtained; then it rose to 100/54 and respiration improved again. While the catheter at the diaphragmatic level controlled the bleeding and maintained a pressure, it also obscured the bleeding points so that they could no longer be found.

After 10 minutes at this level, the balloon was slowly, partially deflated to expose the bleeding points; but the blood pressure was lost again. Re-inflation of the catheter balloon returned the blood pressure to 96/54; but again it obscured the multiple bleeding points.

As the catheter was repeatedly deflated to demonstrate the bleeding points, the blood pressure continued to fall and the patient's condition gradually became worse until he died on the operating table. It was feared that prolonged use of the high aortic tamponade might have resulted in liver, renal, or spinal cord damage from anoxia.

During surgery, the patient received 13 additional pints of whole blood, 1 gm. of calcium gluconate and 1,000 cc. of dextran with 4 cc. of norepinephrine.

At autopsy, no damage to the celiac axis was demonstrated. In addition to the damaged liver and iliac vein, however, there were multiple injuries to branches of the splenic artery and the mesenteric arteries, as well as a severed spermatic artery.

CASE 3. A third patient, also admitted in coma, was treated without early use of the catheter, but without success. This patient had a gunshot wound through the sternum, abdomen, colon, right internal iliac vein, sacrum, and back, as well as multiple small bowel wounds. He was admitted 2.75 hours before surgery, during which time he received 20 pints of whole blood through three portals, one intra-arterially. His abdomen continued to swell and only at one time during resuscitation was there detectable blood pressure and that was a fleeting beat around 40 systolic. When taken to the operating

room, the patient was cyanotic under oxygen, with gasping respiration and unobtainable blood pressure and pulse. The catheter was quickly inserted through a pulseless left common femoral artery and the balloon was inflated at the level of the diaphragm. Blood pressure could not be obtained. Forced respiration was continued and the abdomen was opened almost immediately. Approximately 6,000 cc. of blood were aspirated. A severed, internal iliac vein was controlled; but the patient died almost simultaneously. He had been given 2,000 cc. additional blood while on the operating table.

Discussion

The catheter was not used in moribund patients of the second category, *i. e.*, those who were in shock from causes other than blood loss. All the patients observed in this group had other complicating factors; and it was felt that these complications would not be overcome by the use of the catheter. While prolonged use of the catheter may result in liver, renal, and cord damage, complications from its use are unknown clinically. Edwards and co-workers found that prolonged experimental use of the balloon for periods of aortic occlusion above the celiac and superior mesenteric arteries resulted in irreversible shock in a high percentage of cases.³ While occlusion of the hepatic vessels has received much attention in the literature recently, conceivably occlusion above the celiac axis may still allow some collateral blood flow to the liver. Twenty minutes has been reported as a maximal time, without showing liver necrosis, for continuous occlusion of the hepatic vessels in normal dogs.⁴ Other investigations have reported occlusion of the lower thoracic aorta for 45 minutes without complications.⁵ This finding, however, was during the resection of an aneurysm.

Probably each of these patients reported would have had a better chance for survival if the catheter had been utilized earlier. Fear of harming a patient, who might have been resuscitated without use of the catheter, caused us to reserve it for only moribund patients in whom there was little hope of recovery. It is believed that at least the first patient, and possibly the other two might have been saved if the intra-aortic tamponade had been used earlier. It is felt that, as more experience is gained, the catheter may have a place in the treatment of patients of the first category. It should be evaluated further for such use.

Summary

An intra-aortic balloon catheter tamponade was utilized in two moribund Korean casualties who had uncontrolled, intra-abdominal hemorrhage. Although both patients expired, the catheter was effective in restoring the blood pressure temporarily in one case. The catheter should be evaluated further, both experimentally and clinically.

References

1. Dotter, C. T., and Lukas, D. S.: *Acute cor pulmonale*. Am. J. Physiol. 164: 254, 1951.
2. Sarnoff, S. J.: Personal communication. Harvard School of Public Health, Boston, Massachusetts.
3. Edwards, W. S.; Salter, P. P., Jr., and Carnaggio, V. A.: Intraluminal Aortic Occlusion as a Possible Mechanism for Controlling Massive Intra-Abdominal Hemorrhage. Surgical Forum: 38th Clinical Congress of the American College of Surgeons, 1953, pp. 496-499. W. B. Saunders Company, Philadelphia, 1954.
4. Raffucci, F. L., and Wangenstein, O. H.: Tolerance of Dogs to Occlusion of the Entire Afferent Vascular Inflow to the Liver. Surgical Forum: 38th Clinical Congress of the American College of Surgeons, 1952, pp. 191-195. W. B. Saunders Company, Philadelphia, 1953.
5. De Bakey, M. E., and Cooley, D. A.: Surgical Treatment of Aneurysm of Abdominal Aorta by Resection and Restoration of Continuity with Homograft. Surg., Gynec. & Obst. 97: 257, 1953.

Chapter 4

Evaluation of the Use of a Standard Tilt Test*

Lieutenant Colonel Curtis P. Artz, MC, USA

Not infrequently a patient is admitted to the hospital with an appreciable blood loss and still he appears to be in good condition. Such a patient has been able to compensate for his blood-volume deficiency by vasoconstriction. From the usual clinical signs, he appears to be able to withstand operation; however, when his compensatory ability is abolished by anesthesia, he may have a blood-volume deficiency of 1,000 cc. or more and his blood pressure may fall. When further blood loss occurs during operation, the resulting oligemia may be severe.

In the management of large numbers of casualties in the 46th Surgical Hospital (Mobile Army) in Korea, it was frequently observed that movement of the patient to x-ray abolished compensatory mechanisms. Because of this, it was believed that some type of standard postural change might be of value in determining the circulatory status of an injured patient.

It was the purpose of this study to evaluate the use of a standard tilting procedure in order to estimate blood-volume deficiency and adequacy of preparation for operation.

Methods

A simple tilt table was devised (Fig. 1). The table was made of wood having a platform of convenient size to hold a litter. The platform was suspended with the fulcrum in the center. This type of table enabled a litter patient to be placed on the platform so that his head could be raised or lowered. The degree of tilt could be adjusted as desired.

All patients studied were young, healthy males who had been injured in combat. When a patient's vital signs were approximately normal, he was tilted for varying periods of time with his body elevated to a 30-degree angle, head up. If his blood pressure fell and his condition deteriorated on tilting, it was believed that appreciable blood-volume deficiency was present. All patients were tilted at this same angle, head up. Patients who were put on the tilt table were

*Previously published in *Surgical Forum*, Proceedings of the Fortieth Clinical Congress of the American College of Surgeons, p. 803. W. B. Saunders Company, Philadelphia, 1955.

examined carefully to make sure that hemorrhage had ceased and that physiologic derangements other than blood-volume deficiency had been corrected. The duration of the tilt and the degree of deterioration of the patient's condition were correlated with the amount of blood required to stabilize the patient.

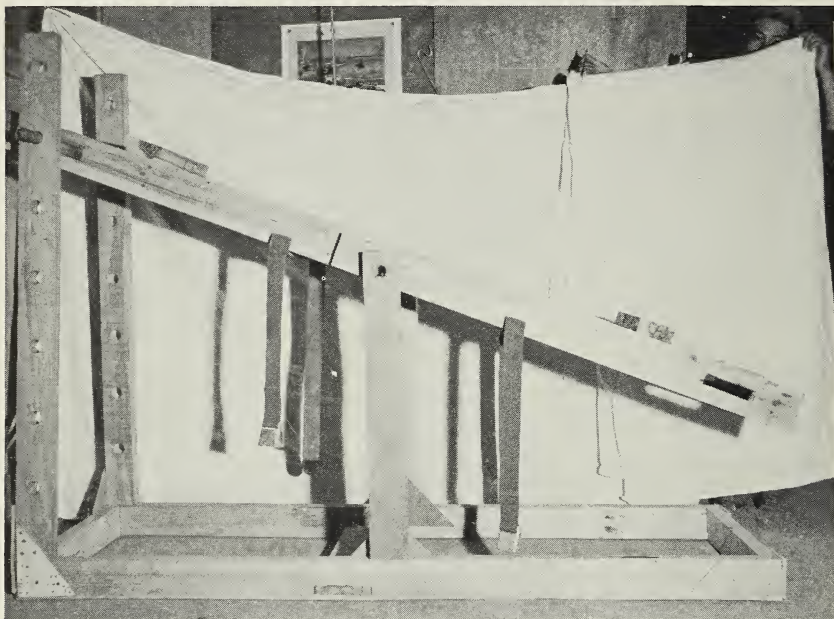


FIGURE 1. A tilt table of wood made by carpenters in Korea. The platform is on a fulcrum. The patient on the litter is placed on the platform.

Results

Several patients admitted to the preoperative section of the forward surgical hospital were placed on the tilt table and their response to tilting was observed. All patients who showed definite deterioration, when placed in the tilt position for several minutes, remained relatively stable when tilted *after* adequate transfusion.

The tilting of a patient aided the surgeon considerably in determining the need for blood.

Seven patients were selected for specific study. All patients showed a significant fall in blood pressure after the initial tilt, but no significant deterioration was observed following adequate transfusion. None of the patients who withstood tilting for 10 minutes or more experienced hypotension at the time of anesthesia. The following brief summaries illustrate salient points.

PATIENT 1 (Fig. 2). An American soldier was admitted with a blood pressure of 120/80, pulse 78, following small penetrating wounds of the upper quadrant of the abdomen and buttocks. His condition appeared excellent. When tilted

Evaluation of Use of a Standard Tilt Test

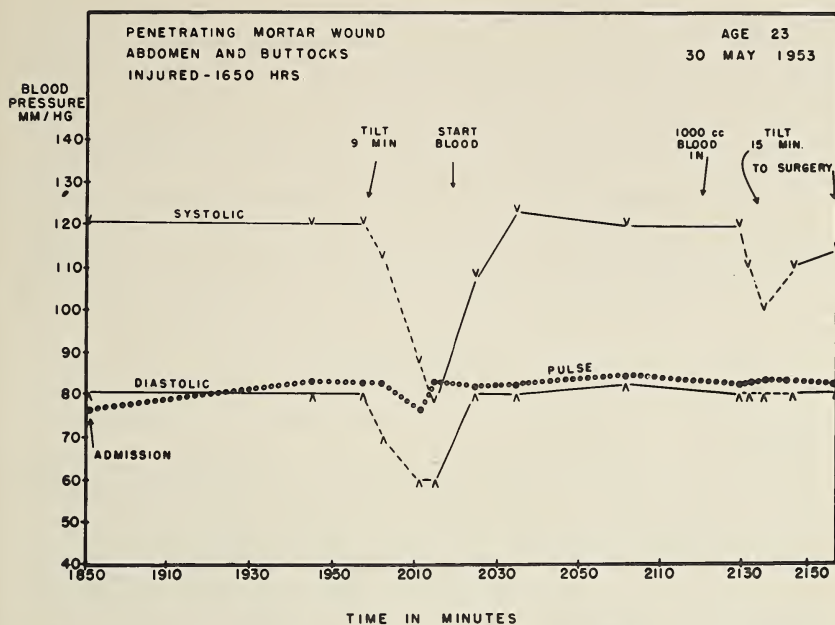


FIGURE 2. Chart of Patient 1. This patient's blood pressure was normal, but his condition deteriorated when he was tilted. Later, after infusion of 1,000 cc. of blood, he withstood tilting well.

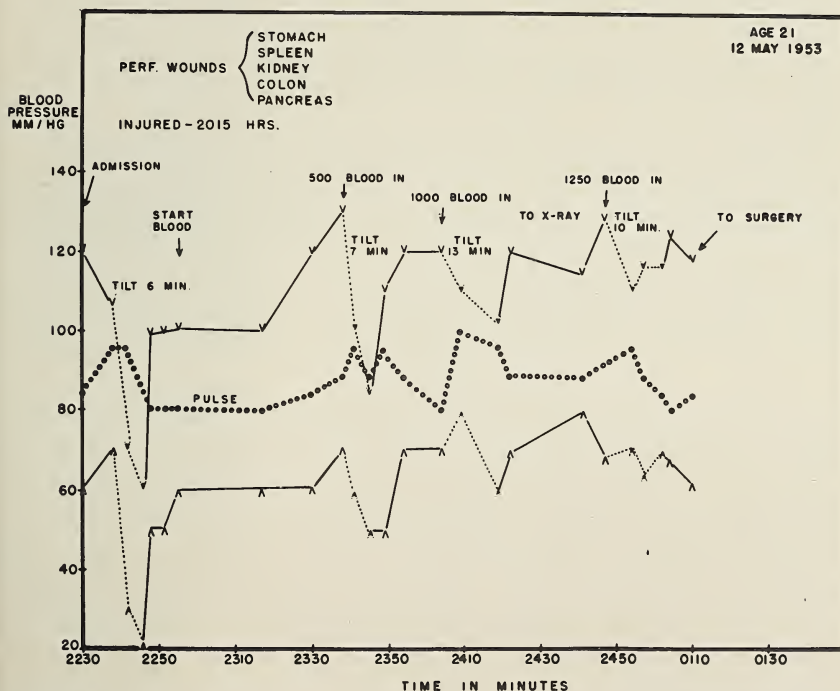


FIGURE 3. Chart of Patient 2. The response to tilting during resuscitation was in direct relation to the amount of blood given.

for 7 minutes, he became nauseated and pale; and sweat broke out on his forehead. His blood pressure fell to 78/60. Following administration of 1,000 cc. of blood, his blood pressure remained stable after tilting. He experienced no difficulty during splenectomy and débridement.

PATIENT 2 (Fig. 3). An American soldier received perforations of the colon, pancreas, kidney, and spleen from small arms fire. He was admitted with a blood pressure of 120/60, pulse 84; but when tilted, his blood pressure fell to 70/30 within 3 minutes. After administering each 500 cc. of blood, the tilt test was performed. At each tilt, blood pressure fell until he had received 1,250 cc. of blood, at which time there was no particular deterioration in his condition. He withstood the extended operative procedure well, in spite of a tremendous blood loss. In the first 24 hours after injury, the total amount of blood received was 14 pints. He experienced no difficulty until 7 days postoperatively, when he developed a severe pancreatitis and peritonitis and died. During his pre-operative resuscitation, this patient was tilted at various intervals; and, in each instance, his response appeared to give a good indication as to the status of his circulation.

PATIENT 3. A Korean soldier was admitted to the hospital with a blood pressure of 120/80, pulse 84, following a penetrating wound of the left side of the chest. He appeared to be in excellent condition. When he was tilted for 5 minutes, his blood pressure fell and cold perspiration broke out on his face. Following infusion of 1,000 cc. of blood, his blood pressure remained stable at 124/76 during a 13-minute tilt. He had no difficulty during the closure of the sucking wound of the chest.

PATIENT 4. A Korean soldier was admitted with a blood pressure of 120/68, pulse 88, a few hours after receiving a perforating wound of the upper abdomen. After tilting, his blood pressure fell to 88/57, pulse 120. His blood pressure rose to 120/60 when lowered to a horizontal position. After infusion of 1,000 cc. of blood, he was tilted again. Following a 15-minute tilt, there was no change in his blood pressure. At operation there were perforations of the liver, duodenum, and transverse colon. He withstood the operative procedure well.

PATIENT 5. An American soldier was admitted with a blood pressure of 110/60 following a grenade wound of the right thigh. After a 5-minute tilt, his blood pressure fell to 80/50, pulse 86. He was pale; and he complained of being sleepy and thirsty. He was shifted to a level position and given 1,000 cc. of blood rapidly. His blood pressure rose to 120/80; and he was tilted for 15 minutes. Blood pressure remained stable. After receiving 75 mg. of demerol intravenously, he was tilted again for 10 minutes. On tilting, no change in vital signs was observed. He withstood a rather extensive débridement of the multiple grenade wounds of the thigh without difficulty.

PATIENT 6. A Korean soldier was admitted with a blood pressure of 70/40, pulse 100, following a land-mine wound of the right lower leg and a penetrating wound of the left upper quadrant of the abdomen from which 24 inches of small bowel protruded. He was pale; and there was a clinical impression of severe oligemia. He received 1,000 cc. of blood during the next hour, and his blood pressure rose to 92/30. On tilting, his blood pressure fell precipitously and his pulse became irregular. He then received a second liter of blood and his blood pressure rose to 124/90, pulse 112. After a 14-minute tilt, no change occurred in blood pressure or pulse. Following an additional infusion of 500 cc. of blood, a prolonged operative procedure was carried out without difficulty. His good response to anesthesia and the operative procedure indicated that he was well resuscitated.

PATIENT 7. A Korean soldier was admitted to the hospital 3 hours after receiving mortar-fragment wounds of the abdomen, shoulder, an open, com-

minuted fracture of the right femur and below-knee traumatic amputation of the right leg. He had an unobtainable blood pressure. Three intravenous infusions and one intra-arterial transfusion were started. After infusing 9 pints of blood, his blood pressure rose to 128/90. It was difficult to determine whether or not this patient had received sufficient blood to withstand operation. He was tilted for 10 minutes; and there was no change in blood pressure or pulse rate. He was given 3,000 cc. of blood and 1,000 cc. of dextran during the prolonged operative procedure. He experienced no difficulty during operation and recovered.

This very severely wounded patient required a vast quantity of blood for resuscitation. The use of the tilt test was of value in determining when adequate blood replacement had been carried out.

Discussion

Observations in this study show that a patient whose blood pressure was within normal limits, but who apparently had lost a fair amount of blood, would have a fall in blood pressure upon tilting his body to a 30-degree angle, head up. Immediately following adequate blood replacement, however, he was able to tolerate the same change in body position without fall in blood pressure.

It is generally believed that, following blood loss, compensation by vasoconstriction is mediated through the sympathetic nervous system. When the body of a hypovolemic patient is tilted, decreased blood to the brain makes for cerebral anoxia. With the loss of central nervous system control, sympathetic activity disappears and the compensatory mechanism is abolished.

Determination of blood volume before operation would be ideal if time, equipment, and space would permit. In most hospitals, this is not only impracticable but almost impossible as a routine procedure. On the other hand, the tilt test is a simple procedure. It can be utilized rapidly, without discomfort to the patient and without critical equipment. Although no blood-volume determinations were performed in correlation with responses to the tilt test, there was clinical evidence during the operative and postoperative period of the patient's stable response to a 10-minute tilting. This proved to be a good indication of adequate replacement.

These observations are in agreement with those of Green and Metheny, who studied estimation of acute blood loss by the tilt test. They felt that in the absence of a syncopal reaction, an increase in cardiac rate less than 25 beats per minute on tilting indicated either a negligible or a compensated acute blood loss. Transfusion was not required unless an increase in cardiac rate of 30 beats or more per minute was observed on tilting. This suggested a blood-volume deficiency of from 9 to 14 cc. per kilogram of the patient's body weight and a probable transfusion requirement of 1,000 cc. of blood. These investigators felt that the occurrence of a syncopal reaction to tilting

suggested a probable blood-volume deficiency of 1,500 cc. of blood.¹

Duncan, Sarnoff, and Rhode studied the effect of postural change in 30 patients with varying degrees of injury. They noticed that less severely injured patients who were placed in a head-up position experienced a state of collapse. They believed that this was due to vasodilation and pooling of blood in dependent areas and resultant cerebral anemia.²

One of the most perplexing problems in patients who have experienced varying degrees of blood loss is the determination of adequacy of replacement. From the size and character of a wound, experienced surgeons can estimate the approximate amount of blood required before surgery can be attempted. Great difficulty is encountered, however, in determining the adequacy of resuscitation in the very severely wounded patient. Without adequate knowledge of the amount of blood lost, it is difficult to determine the state of resuscitation *after* a patient has received 6 or 7 pints of blood. Tilting the body of a patient at a 30-degree angle, with head up, seems to be a test of additional value in determining the adequacy of the circulation.

Summary

Several wounded patients whose circulatory status was difficult to determine by clinical observation were placed on a tilt table and their bodies were tilted to a 30-degree angle, head up. If the blood pressure fell and the patient showed other signs of deterioration, it was believed that further transfusions were required prior to operation. When the patient's condition remained stable during a 10-minute tilt, it was believed that adequate replacement therapy had been carried out. Although the experience with the tilt table has not been extensive enough to formulate definite policies concerning its routine use, careful studies in several patients suggested that the response to tilting offers additional criteria for determining the status of the circulation and adequacy of replacement therapy.

References

1. Green, D. M., and Metheny, D.: The Estimation of Acute Blood Loss by the Tilt Test. *Surg., Gynec. & Obst.* 84: 1045, 1947.
2. Duncan, G. W.; Sarnoff, S. J., and Rhode, C. M.: Studies on the Effects of Posture in Shock and Injury. *Ann. Surg.* 120: 24, 1944.

Chapter 5

Experiences With Intra-Arterial and Rapid Intravenous Transfusions in a Forward Surgical Hospital*

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Captain Yoshio Sako, MC, USA

Captain Alvin W. Bronwell, MC, USA

Early in the Korean conflict, intra-arterial blood transfusion was used in the resuscitation of hundreds of men critically injured in battle. The resulting impressions of the many surgeons in the theater varied so widely that, after an extensive clinical experience, we initiated a clinical evaluation of transfusion by the intra-arterial route in an attempt to outline the possible indications for the procedure. During the early phase of the study, every patient with severe oligemic shock received an intra-arterial transfusion. Blood was usually transfused into the femoral artery through a 15- or 17-gauge needle. The femoral artery was surgically isolated and the surgeon held the needle in the artery during the administration of the blood. A Rochester plastic needle was used in two patients after cutting down on the femoral artery, and in one patient blood was given directly into the aorta through a 17-gauge needle during a laparotomy.

The technic of surgically isolating the femoral artery and holding the needle in place during the rapid infusion of blood was feasible. Minimal amounts of blood were given simultaneously by intravenous infusion. The maximum amount of blood normally given intra-arterially was 3,000 cc. although in one patient who received 4,500 cc., intra-arterial transfusion was discontinued as soon as the patient's systolic pressure reached 100 and further replacement therapy was given intravenously. No complications occurred from this therapy. When the needle was removed from the artery, any bleeding was readily controlled with a small piece of gelfoam and gentle pressure. The large-gauge needle, because of its larger lumen, was preferable to the Rochester plastic needle. Unless the surgeon steadied the needle during the pressure transfusion, it would impinge on the wall of the artery and a free flow of blood would be impeded.

Results

After seven patients in severe oligemic shock had been resuscitated by intravenous blood transfusion through multiple veins, the data were

*Previously published in *U. S. Armed Forces Medical Journal* 6: 313, 1955.

Table 1. Patients Receiving Intra-Arterial Blood Transfusions

Pa- tient	Wound	Blood Pressure on Ad- mission	Time in Min- utes	Amount of Blood (cc.)	Rate per Min- ute (cc.)	Results
1	Multiple, of body; sucking, of chest; traumatic amputation of right hand	0/0	45	3,000	70	Recovered
2	Traumatic amputation of left leg; penetration of right thigh, abdomen, and hand; fracture of clavicle		60	4,500	75	Recovered
3	Traumatic amputation of right leg; fracture of radius and humerus; perforation of chest	0/0	35	3,000	86	Died on operating table
4	Penetration of chest and abdomen; perforation of right arm	0/0	30	1,500	50	Recovered
5	Perforation of inferior vena cava; laceration of left kidney; perforation of duodenum	120/80	10	1,500	150	Died 3.5 hours following operation
6	Penetration of abdomen and sigmoid colon; laceration of superficial femoral artery; penetration of right leg	0/0	30	3,000 (and 1,500 cc. dextran)	100	Recovered
	Average rate				88	

Table 2. Patients Receiving Rapid Intravenous Blood Transfusions

Patient	Wound	Blood Pressure on Admission	Time in Minutes	Amount of Blood (cc.)	Rate per Minute (cc.)	Results
1	Penetration of abdomen and legs; laceration of common iliac artery; retroperitoneal hematoma	0/0	45	3,000	70	Died on operating table
2	Penetration of thigh; laceration of superficial artery	140/70	60	5,000	83	Recovered
3	Multiple penetrations of right buttocks; open fractures of sacrum; penetration of rectum; traumatic amputation of right leg	50/30	108	5,000	46	Died
4	Penetration of abdomen and right leg	140/80	15	1,500	100	Recovered
5	Penetration of thorax and abdomen; laceration of spleen	80/40	45	3,000	70	Recovered
6	Traumatic amputation of left thigh; multiple, right leg, right thigh, and left arm	30/?	75	4,000	53	Recovered
	Traumatic amputation of both legs and left hand	90/70	65	5,000	77	Died on first postoperative day
	Average rate				70	

assembled. Six patients had received intra-arterial transfusions and seven had received rapid intravenous infusions. In the clinical evaluation of the patients in the two groups, the degree of shock was comparable. Four patients in the former group and only one in the latter group were admitted with an unobtainable blood pressure. The degree of injury and amount of hemorrhage, however, was greater in those receiving blood intravenously, and the amount of blood required for resuscitation was slightly higher. The data on these patients are summarized in Tables 1 and 2.

There was no appreciable difference in the rate at which blood was given to patients; via artery it was 88 cc. per minute and via vein, 70 cc. per minute. No attempt was made to achieve an absolute maximum rate of infusion by either method. These data represented the actual accomplishments of blood replacement at one forward surgical hospital in a small, comparable series of patients in deep oligemic shock. In one moribund patient not included in this series, a definite attempt was made to determine how rapidly blood could be infused by the intravenous route. In 30 minutes 5,500 cc. of blood were injected into two veins through 15-gauge needles; 3,500 cc. of the blood were pumped into one vein in 21 minutes. This demonstrated that blood can be given very rapidly by the intravenous route.

After the controlled use of intra-arterial transfusion in six patients, it was our belief that the patients did not show any appreciably improved response as compared with patients who received blood at a comparable rate through multiple, intravenous routes.

Discussion

Proponents of the belief that intra-arterial transfusion is superior to intravenous transfusion in the treatment of hemorrhagic shock have cited the following reasons to account for its supposed superiority: (1) blood given intra-arterially increases the coronary and cerebral arterial flow; (2) direct infusion into the arterial tree causes an instantaneous rise due to the simple hydrostatic effect; (3) blood given intravenously would tend to pool, whereas intra-arterial blood readily mixes; and (4) blood can be administered more rapidly by the intra-arterial route.¹ These four reasons are given as prime factors in the superiority of intra-arterial transfusion over intravenous infusion. At the time most of these statements were made, there was no experimental or clinical evidence to support their contentions.

Recently a number of well-designed experiments were carried out in an attempt to evaluate the role of some of the above-mentioned factors. Kohlstaedt and Page,¹ and Case and associates,² studied the effects on coronary flow and arterial pressure following intra-arterial and intravenous infusion of equal magnitude in dogs in oligemic

shock for varying periods of time. They were able to show in repeated experiments that coronary flow and arterial pressures responded just as rapidly and to the same extent with either intravenous or intra-arterial blood transfusions. In addition, the right and left auricular and pulmonary artery pressures were measured and found to be the same during both types of transfusion. Their conclusion was that their data did not constitute a contraindication to intra-arterial blood transfusion, but that there was no convincing evidence that it was superior to intravenous blood transfusion.

Patients in shock may not have a discernible vein suitable for giving infusions, but if by surgical means the same search is made to locate a vein as an artery, it could be found. Moreover, when necessary, the femoral vein is always available for cannulation. The statement that blood can be administered more rapidly by the intra-arterial than by the intravenous route is not true. In summarizing their findings, Case and associates stated that the rate of administration is a function of the pressure in the bottle, pressure in the vessel, resistance to the flow of blood in the needle and tubing, and the viscosity of blood. Under these circumstances, blood can be administered more rapidly through the intravenous route.

Another statement frequently made is that blood administered by the intravenous route will produce cardiac failure. There is evidence to show that this is so only after the blood pressure has returned to normal or when there is an underlying cardiac disease. The contention that direct infusion into the artery at rates as currently practiced will cause a rise in pressure due to hydrostatic effect has not been substantiated.

Maloney and co-workers³ gave rapid infusions of blood, both intravenously and intra-arterially, and measured cardiac output by the blue-dye injection method. They also measured mean arterial pressure on a series of normal dogs and on dogs in shock due to hemorrhage; they were unable to demonstrate any difference in the rise in arterial pressure by transfusion through either route. Case and associates cited other experimental work along the same line of investigation indicating that an animal in oligemic shock did not respond more favorably to intra-arterial blood transfusion than to intravenous blood transfusion. The volumes of blood concerned when transposed from the rate of a dog to that of a man were in the realm of 445 cc. per minute; this rate is much higher than the rates given in a clinical operation.

Richards and Hansen⁴ also studied the comparative action of intra-arterial and intravenous transfusion in the treatment of oligemic shock. They found no demonstrable advantage of intra-arterial over intravenous transfusion when equal infusion rates were used. They stated that rapid intravenous transfusion was well tolerated, particu-

larly when vessels were in a collapsed state due to hemorrhage. They concluded that this was logical because the blood flow to and from the heart per minute is many times the volume of blood which can be forced into the circulation by any type of pressure of transfusion in a similar length of time. Prior to their experimental work, statements regarding the advantages of intra-arterial transfusion over the intravenous route were made without being substantiated by clinical or laboratory experiments.

A situation in which intra-arterial transfusion may be indicated is in the resuscitation of a patient whose heart is in asystole because, once the output of blood from the left ventricle has ceased, there is no other way for the blood to get into the arterial tree. Another situation would be the sudden need for massive transfusion during an operation for mitral stenosis to get blood into the arterial tree. Except for these two unusual circumstances, most of us believe that there are certain disadvantages associated with the process of intra-arterial transfusion. Cases have been reported of arterial insufficiency necessitating amputation of an extremity,⁵ tissue necrosis, arterial spasm caused by arterial cannulation, and in some instances a delay in getting the blood infused because of additional time required to begin the intra-arterial transfusion. A major criticism by most investigators on the work of those who present the advantages of intra-arterial transfusion over intravenous transfusion is that their clinical as well as experimental investigations have not dealt with comparable rates of infusion of blood.^{2 4} In some instances conclusions were based on patients in whom four times as much blood was given by the intra-arterial route as was given to others by the intravenous route.

Because of the impressions gained from patients in whom blood was given primarily by the intra-arterial route coincided clinically with the above experimental data, intra-arterial transfusions were discontinued in favor of rapid intravenous infusion of blood through multiple, large-gauge needles or intravenous cannulae.

Conclusions

No definite conclusions can be drawn from this very limited experience. Our impressions were that blood given by the intra-arterial route was of no more value in the resuscitation of patients in deep oligemic shock than was blood administered by the intravenous route, if it was given at the same rate. Observations in one forward surgical hospital showed that, in a small series of severely wounded patients of comparable severity, blood replacement was accomplished as rapidly by multiple intravenous routes as it was by the intra-arterial route.

Summary

The clinical evaluation of intra-arterial blood transfusions administered to six patients who were wounded in combat was compared to the results obtained by rapid intravenous transfusions given to seven men who were also combat casualties. The average rate of blood given intra-arterially was 88 cc. per minute, and intravenously, 70 cc. per minute. These were actual rates at which blood was given and not the maximum rates possible. There was no discernible difference in the rate of response between the patients who received blood intra-arterially and intravenously.

References

1. Kohlstaedt, K. G., and Page, I. H.: Hemorrhagic Hypotension and Its Treatment by Intra-arterial and Intravenous Infusion of Blood. *Arch. Surg.* 47: 178, 1943.
2. Case, R. B.; Sarnoff, S. J.; Waithe, P. E., and Sarnoff, L. C.: Intra-arterial and Intravenous Blood Infusion in Hemorrhagic Shock. *J. A. M. A.* 152: 208 (May 16), 1953.
3. Maloney, J. V., Jr.; Smythe, C. McC.; Gilmore, J. P., and Handford, S. W.: Intra-arterial and Intravenous Transfusion: Controlled Study of Their Effectiveness in Treatment of Experimental Hemorrhagic Shock. *Surg. Gynec. & Obst.* 97: 529, 1953.
4. Richards, R. C., and Hansen, F. L.: Comparison of Intra-arterial and Intravenous Transfusion in Treatment of Hemorrhagic Shock. *Surgical Forum*, Proceedings of the Forum Sessions, pp. 478-484, Thirty-ninth Clinical Congress of the American College of Surgeons, Chicago, Illinois, 1953. W. B. Saunders Co., Philadelphia, Pa., 1954.
5. Ziperman, H. H.: Personal communication.

Chapter 6

Massive Transfusion in the Severely Wounded*

Report of a Case Receiving 23,350 cc. of Blood
in the First Twenty-four Hours

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The experiences of World War II pointed out the value of whole blood in the management of the severely wounded. During the Korean conflict, it became apparent that large amounts of blood should be administered to the battle casualty. Replacement of an amount equal to twice the blood volume was not uncommon. As time went on, reports came from various hospitals of the tremendous amounts of blood given to some casualties. The case herein reported received 23,350 cc. of stored, type-O, low-titer blood in the first 24 hours after injury.

Case Report

A 22-year-old American airman was injured by mortar-shell fragments on June 26, 1953, at 1145 hours. He sustained a penetrating wound of the right upper quadrant of the abdomen, a perforating wound of the left hip and right leg. He arrived at the clearing station 20 minutes after injury. At that time his blood pressure was 0/0 and he was cold, clammy, and semiconscious. Blood transfusion was started 10 minutes later; and, in the next 30 minutes, 1,700 cc. of blood were administered. The patient's color improved; he became responsive; and a blood pressure of 130/? was obtained. He was then moved to a surgical hospital by ambulance, arriving 2.67 hours after injury.

Upon admission, his blood pressure was 140/80 and his pulse rate 120. Thirty minutes after admission, 1,000 cc. additional blood had been administered through a cannula in the left leg; but blood pressure fell to 90/48, pulse rate 142, respiration 42. Transfusion was continued during the time roentgenograms were taken. There was a metallic fragment in the right lower quadrant at the level of S-I. There was an open fracture of the body of the left ilium, with metallic fragments lying lateral to the greater trochanter and an open fracture of the upper portion of the right tibia.

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A catheter was inserted into the bladder and the urine was found to be clear. Since there was a fall in blood pressure and a pulse rate of 160, in spite of the infusion of blood, it was believed there was continued intra-abdominal hemorrhage. There was progressive rigidity and distention of the abdomen. An additional cannula was inserted into a vein in the left arm since vena cava injury was suspected. The patient was taken to surgery 4.75 hours after injury. He had received 5,700 cc. of blood at this time; but his blood pressure was 66/0.

After pentothal induction and endotracheal ether anesthesia, an exploratory laparotomy was performed through a right rectus incision. Upon incision of the bulging peritoneum, approximately 3,000 cc. of blood was found. The abdomen was rapidly explored, beginning with the iliac region. Pressure was then placed in the region of the celiac axis and the entire area was observed for bleeding points. A 7-inch, through-and-through tear of the inferior half of the right lobe of the liver was bleeding. A gauze pack was inserted; and it appeared that the hemorrhage was under control. Until 2,000 cc. additional blood was administered under pressure, further surgery was withheld. The patient's blood pressure rose to 100/64; and his pulse rate was 120. Although gelfoam was placed in the laceration of the liver, bleeding did not cease. Through-and-through sutures of chromic catgut (No. 1) in round, curved needles were placed widely in the liver to pull the laceration against the gelfoam pack. Finally hemorrhage was controlled by placing two figure-of-eight sutures with gelfoam under the structure on the anterior and posterior aspects of the liver.

The abdomen was explored thoroughly, and no further intraperitoneal damage was observed. The cecum and ascending colon were reflected medially; and the missile tract in the right psoas muscle was located. The muscle was débrided; a 2 cc.-square, metallic fragment was removed; and the bleeding points were ligated. Four drains were placed about the liver and brought out through a stab wound in the right flank. Two large drains were placed in the retroperitoneal area, around the damaged psoas muscle, and brought out through a separate stab wound. A final examination showed that bleeding was under control. The abdominal wall was closed in layers. The wound of entrance to the abdomen and wounds of the extremities were débrided. During the operation which lasted 1.25 hours the blood pressure was around 100/60 for approximately half of the time; and it went as low as 60/0 at other times. During the operation, the patient received a total of 4,500 cc. of blood, making a total of 20 pints administered following injury. He left surgery with a blood pressure of 80/60, pulse rate 120, and respiration 40.

For the first few hours postoperatively, the patient's pulse rate increased to 150; and his blood pressure fluctuated between 80 and 100

(systolic) in spite of transfusion of an additional 2,000 cc. of blood. During the first 3 hours after surgery, he excreted only 7 cc. of urine. A great deal of oozing could be seen from the wounds, and considerable loss of blood was occurring through the two drainage sites. Three gm. of calcium gluconate were given intravenously with no noticeable effect on the oozing or blood pressure.

Four hours after surgery, the patient's pulse continued to be 150 and his blood pressure 90/68. A total of 3,500 cc. of blood had been administered following operation, making a total of 27 pints since injury. It was believed that this patient continued to ooze from all wound surfaces, and possibly from the liver. A secondary exploration was thought advisable, inasmuch as the administration of 875 cc. of blood each hour did not compensate for the blood loss.

The patient was taken to the operating room again 11 hours after injury. His blood pressure was 50/40, and his pulse 130. An intra-arterial transfusion was started in the right femoral artery and blood was also pumped into the arm and leg cannulae. The abdomen was re-entered through the previous incision and a massive amount of blood was found in the peritoneal cavity. The liver laceration was not bleeding. A large amount of blood was seen in the depths of the right psoas muscle wound and retroperitoneal area. No definite bleeding point could be found. A large gelfoam pack and two 5-yard gauze packs were inserted into the retroperitoneal area and the ends brought out through an incision in the right flank. Four drains were again placed around the liver. The abdomen was closed with through-and-through wire sutures.

Twelve pints of blood were given during the 1-hour operation, three of them into the femoral artery, making a total of 39 pints. During the operation, the blood pressure was 60/40 and, after administration of 1 ampule (4 mg.) of norepinephrine, the blood pressure rose to 110/80. With discontinuance of norepinephrine, the patient's pulse was 140 and his blood pressure was 0/0. An additional 4 gm. of calcium gluconate were given with no discernible effect.

After the completion of the operation, the patient remained on the operating table under the care of the anesthesiologist for some time. The patient continued to ooze; and it was decided that freshly drawn blood might be of value in stopping the oozing. By 0200 hours, 40 pints of blood had been given (1 pint of fresh blood); the patient's pulse was 140, respiration 48, blood pressure 110/70. Vitamin K-1 oxide (100 mg.) was given intravenously and 3 additional pints of freshly drawn blood were given slowly. It was estimated that the patient continued to ooze at the rate of approximately 300 cc. per hour. Following the operation and administration of 43 pints of blood (4 of which were freshly drawn), the output of urine was 150 cc. on the third hour and approximately 70 cc. per hour thereafter. At this time the patient's general condition appeared to be good.

At 0500 hours, the patient experienced an acute gastric distention because of malfunction of an indwelling stomach tube and respiration increased to more than 60 per minute. This was relieved by the introduction of a larger stomach tube and the respiratory rate fell immediately to 40.

Between the 24th and 30th postoperative hours, the patient improved remarkably; he appeared to have stopped bleeding. Blood pressure was 120/40, pulse 120, respiration 40. Output of urine was 60 cc. per hour. He had received a total of 46 pints of blood (7 pints of freshly drawn blood) and 2,600 cc. of 5 per cent glucose in water within 24 hours following injury.

During the early postoperative period, the patient's course was somewhat stormy. His temperature rose to 103° F. on the first day and slowly fell to normal by the fourth day. He received one million units of penicillin every 4 hours and 1 gm. of Terramycin every 12 hours intravenously. One-half of the pack was removed on the third postoperative day and the remainder on the fourth day. Although there was a great deal of drainage, there was no bleeding. He was sitting in a chair and eating well by the tenth postoperative day, at which time he was evacuated to the 25th Station Hospital.

In a follow-up letter from the Osaka Army Hospital in Japan, it was stated that the patient was admitted on the 12th postoperative day in surprisingly good condition. His hematocrit was 39, total proteins 6.7 gm., albumin 3.2, globulin 3.5. On the 23rd postoperative day, secondary closure of soft-tissue wounds of the leg was carried out. By the 37th postoperative day, he was walking about and was feeling quite well. He had occasional bouts of distention and diarrhea; but it was felt that he was ready for transfer to the United States.

Discussion

After a long evacuation by litter jeep, this severely wounded casualty arrived at the clearing station with a blood pressure that was too low to be obtained by the cuff method. After administration of 1,700 cc. of blood, the patient improved and withstood further evacuation to a surgical hospital. This demonstrates the value of resuscitative measures at the division level. It is wise to delay transportation of a severely wounded soldier for infusion of resuscitative fluids and thus prepare him for further transport, rather than to rush him to a hospital in extremely poor condition.

Although the patient's blood pressure on admission to the hospital was 140/80, it soon fell to 90/48 in spite of continuous infusion of blood. Failure of the patient to maintain a normal blood pressure and a rapidly expanding abdomen were evidences of profuse hemorrhage intra-abdominally.

Since vena cava injury was suspected, an additional cannula for administration of resuscitative fluids was inserted into an arm vein because leg cannulae are of little value in the presence of such injuries.

An operation was carried out immediately in order to control hemorrhage. At operation, a large amount of blood came from the damaged retroperitoneal muscles. Blood loss from this area is always much greater than is apparent. After the brisk hemorrhage was controlled by gauze packs, further operation was delayed until additional blood had been given. This illustrates an important point in resuscitation, namely, when a surgeon suspects continued, profuse, intra-abdominal hemorrhage, it is an indication for immediate exploration. As soon as the hemorrhage is controlled, further manipulation should be delayed until the patient's condition is improved by further replacement of blood.

Although continued oozing was obvious after operation in spite of administration of 876 cc. of blood per hour, hypotension with the accompanying low output of urine was further indication of serious hemorrhage. The second operative procedure was started when the patient was in very poor condition. Blood pressure was 50/0. The patient was reopened under minimal anesthesia; and further restoration with blood was carried out. In this instance, it is doubtful if the intra-arterial transfusion was of particular value because of the slow rate at which it was given—1,500 cc. in 1 hour.

At the first operation, the liver hemorrhage was controlled by large sutures over gelfoam. Bleeding occurred from multiple points in the damaged retroperitoneal muscle. It is not uncommon for oozing to occur after massive transfusion of stored blood. This is believed to be due to a deficiency in the clotting mechanism, the exact nature of which has not been determined. Administration of multiple, large doses of calcium gluconate or vitamin K-1 oxide was of no apparent benefit. Previous experience has established the beneficial effects of fresh blood in patients who have a bleeding tendency after the administration of large amounts of stored blood. In this patient, it is believed that the use of fresh blood was lifesaving. However, a large quantity of blood (7 pints) was required before there was evidence that clotting occurred.

In the postoperative period, nasal oxygen was administered. About 3 hours after operation, respiration increased to 60 per minute. An intragastric tube was in place, but not functioning; and acute gastric distention occurred. This is not an uncommon occurrence in severely wounded patients. A larger tube was inserted; the distention was relieved; and the respiratory rate decreased to 40 per minute.

Postoperatively the patient made a rapid recovery. The pack was partially removed on the second day, and the remainder on the fourth day without further bleeding.

This case emphasizes the value of replacement of blood loss with whole blood.

Summary

The use of massive quantities of blood may be required in the management of severely wounded patients. A case is reported in which a patient received 23,350 cc. of blood in the first 24 hours after injury. Discussion includes several interesting factors in the initial care of patients who have sustained severe injury, accompanied by unusually great blood loss.

Addendum

In a follow-up letter it was learned that this patient developed a subcutaneous evisceration while waiting to be evacuated to the Zone of Interior. At operation, the bowel was inadvertently entered and he developed a jejunal fistula. From then on, his course was steadily downhill and he expired on the 76th day following injury.

Postmortem examination revealed jejunal fistula, peritonitis, bronchopneumonia and cerebrospinal leptomeningitis.

Chapter 7

Resuscitation of the Battle Casualty: A Résumé

Captain John M. Howard, MC, USAR

Section I. Injury and Wound Surgery

The battle wound is a dynamic injury, not a static one. It continues to injure the body for a period of days or weeks, and not simply for a period of hours. As the result of the continuing, deleterious effect of major injury, the body responds *in toto*. Every system, every organ, and presumably every cell in the body, respond to major trauma. This response is not a response of the moment, but a continuing response for days and weeks. The older concept, that the injury was a momentary insult and that an injured man either lived or died as a result of the initial insult, is incorrect. Under this concept, since the injury was a thing of the past, resuscitation could be directed only at its systematic effect. Such a concept is untenable today. The wound is dynamic—past, present, and future—and resuscitation must first include efforts to minimize or reverse the element of continued insult and then include efforts to correct its previous damage. First stop the injury; and then correct its previous damage. This basic concept was developed by Trueta in the Spanish Civil War and later by the surgeons of World War II.

What is the nature of the injury from which one resuscitates the casualty? There are at least four components to the battle injury, any of which may predominate or prove fatal, if left untreated. These are: (1) destruction of tissue, (2) loss of blood, (3) breaks in the defense against bacteria, and (4) occurrence of mechanical defects which may threaten life, limb, or function. A fifth, which ultimately may prove of importance, is the direct transmission of energy from the missile to the entire body, depicted only locally as tissue destruction. Functional derangements, far removed from the missile, may be caused by the direct transmission of energy just as is the case of an electric shock.

The first four components must be corrected, for any one of these may produce a progressive injury. Blood, plasma, and electrolytes are lost continuously from the wound or into the injured tissue. Products of tissue degeneration and bacterial toxins are continuously absorbed from the wound. This new element in the circulation may be working to the net disadvantage of the body. One of the major aims

in the preparation of the casualty is to minimize the effects of the wound by surgical excision. The above points are illustrated in the following outline:

The Battle Wound

1. The battle wound is dynamic. The battle wound results in a defect which produces a continuing deleterious effect. This continuing deleterious effect must be minimized by operative débridement.
2. Following injury the body responds to correct the defects. This is a continuing response of every organ and system which has been studied. This response may be life saving.
3. Anesthesia blocks part of the patient's response and therefore, at least for the moment, furthers the injury.

The response, like the injury, is continuous. Its magnitude and duration appear to be proportional to the magnitude of injury.

The outline of the relationship of a specific response to a specific element of injury is an oversimplification; and it is used here only to demonstrate a philosophical approach to the problem. Under each specific element of injury is listed a variety of specific responses.

Tissue destruction: (1) pain and apprehension, (2) local vascular constriction and retraction, (3) inflammation, (4) shifts of water and electrolytes, (5) paralytic ileus, (6) metabolic débridement of the wound (internal débridement), (7) tissue slough (external débridement), and (8) wound healing.

Blood loss: (1) the response of the autonomic nervous system, (2) increased cardiac activity, (3) the adrenal cortical response, (4) renal vasoconstriction, (5) increased production of red blood cells, (6) increased production of plasma proteins, and (7) increased production of clotting factors.

Bacterial contamination: (1) antibody formation, and (2) leukocytic response.

Mechanical defects: (1) increased cardiac or respiratory activity to overcome effects of specific defects, and (2) muscle splinting.

Nature has effective means of controlling hemorrhage, replacing blood volume, elevating blood pressure, resisting infection, and débriding dead tissue. Yet nature's methods may be inadequate to meet the needs of the critically injured man. It is therefore necessary to recognize the major components of injury and the major deficiencies in response, to correct the former, and to augment the latter. The over-all problem is summarized in Figure 1.

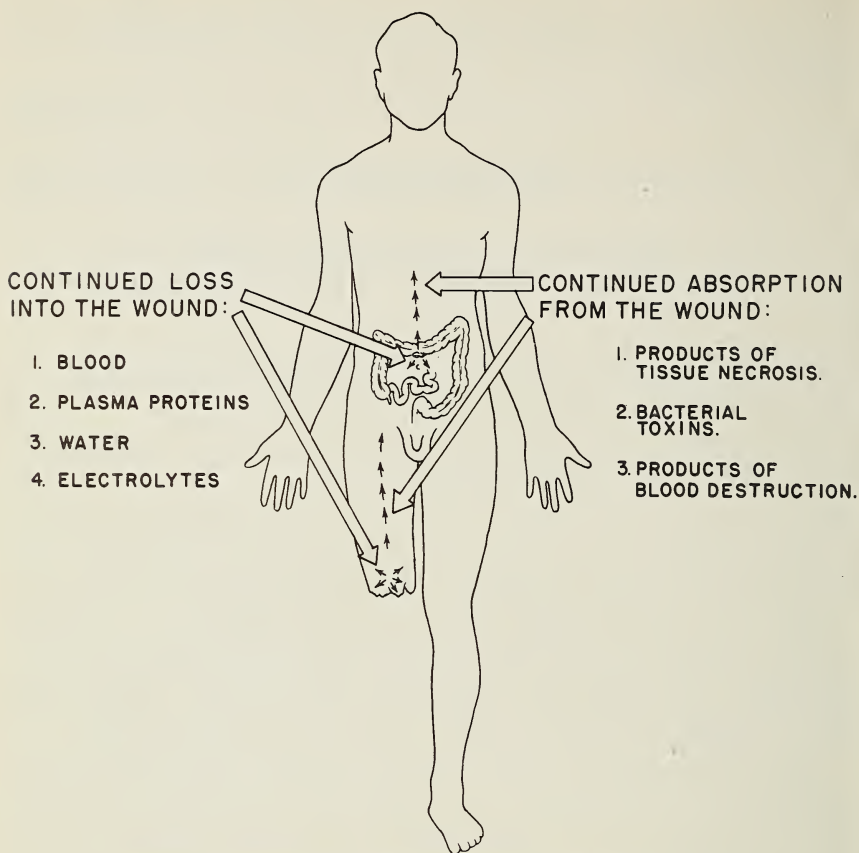


FIGURE 1. Schematic diagram of dynamic effects of injury.

The Local Wound

In one sense, the magnitude of a wound is determined by the amount of energy transmitted from the wounding agent to the body. This transmission of energy is dependent, quantitatively, upon the mass of the wounding agent, its velocity, and the resistance offered by the body.

The most massive wounds encountered in military surgery are the traumatic amputations which occur in land-mine warfare. Clinical experience has clearly indicated that a traumatic amputation of the foot at the ankle is not nearly so severe an injury as a traumatic amputation of the mid-thigh. The force required to amputate a foot at an ankle is presumably less than the force required to sever the femur and its surrounding muscles. Following transfusion, the casualty with the wound at the ankle responds rapidly in a characteristic manner, whereas the casualty with the femoral wound responds

slowly. This is a fact which may be due partially to differences in hemostasis.

The extent of tissue injury resulting from a bilateral traumatic amputation probably includes every cell in the body. The force applied to the lower extremities is severe enough to sever the extremities. Tissue adjacent to the line of amputation is nonviable. Tissue somewhat further away may be nonfunctional but viable. The changes may be reversible. The energy transmission does not stop at this level, however, as the man may be hurled several yards away. Every cell in the body may prove to be affected somewhat like an electrical charge. Such a concept may explain the mechanism of death in some of the men who are killed instantly.

By the same logic, an open fracture is a more severe injury than a simple, soft-tissue injury, as a greater wounding force was expended in producing it.

Working with small, high-velocity missiles, investigators at the Army Chemical Center demonstrated the large cavity of destruction which results from the indirect transmission of energy as a missile passes through tissue. This cavity persists for a fraction of a second only; but the damage to the tissue persists much longer. By this same mechanism, dirt, bacteria, fragments of cloth, and fragments of detached tissue may be scattered over a wide area. Frequently the tissue damage due to this indirect injury may be detectable clinically.

The following injuries have been noted as a result of relatively low-velocity, small-arms missiles:

1. A small wound of entrance in the right upper quadrant of the abdomen and a small wound of exit in the back. The wound of the liver was the size of a double fist.

2. A small perforating wound of the arm, complicated by loss of function of the median nerve. Exploration of the wound revealed no evidence of direct injury to the nerve—an observation made rather frequently.

3. A penetrating wound of the lower thigh, with a cold pulseless foot. Exploration of the wound revealed spasm of the femoral artery without evidence that the vessel had been touched by the bullet.

4. A tangential wound of the chest wall, with the pathway of the missile lying external to a rib. Films revealed a massive hematoma of the lung.

5. Wounds of entrance and exit, each 1.0 cm. in diameter in the calf; and a wound of the gastrocnemius muscle the size of one's fist. Such soft-tissue wounds have been seen repeatedly.

6. A perforating wound adjacent to the vertebral column, without radiological, operative, or autopsy evidence of vertebral injury; yet the patient developed a delayed paralysis of the spinal cord.

The wounds of entrance and exit, as indicated above, do not necessarily depict the degree of injury lateral to the missile's tract; nor do they depict the magnitude of tissue destruction in the underlying tissues.

Physiologic Characteristics of the Wound

Although the production of a wound by the transmission of energy is instantaneous, the effects of the wound become dynamic.

Blood loss continues into the tissues for a variable period of time. Other investigators have demonstrated the loss of albumen into traumatized tissues. Moreover, for an unknown period of time, the loss of blood and plasma is a continuing loss.

Green and Stoner described evidence of the possible toxicity of absorbed products of muscle degeneration. Such toxins from tissue breakdown, as well as toxins from bacterial proliferation, may have a systemic effect. Creatine may be excreted in large amounts after severe extremity wounds. These changes are continuing processes. The continued presence of nonviable tissue has a profound effect on the systemic response to injury, and undoubtedly it represents a continuing injury of major importance. Conversely, radical débridement may be followed by a rapid subsidence of the systemic reaction.

The continued metabolic response imposed by the wound is an additional factor which makes the wound function similarly to a vital organ.

The injury has been discussed as an injury of the entire body, with emphasis on the local tissue destruction. It should also be emphasized that certain wounds specifically affect the function of an entire system. For example, a wound of the jejunum results in a paralytic ileus of the entire gastrointestinal tract. An open wound of the chest affects the efficiency of the entire respiratory system. A wound of the brain may specifically affect the function of the entire nervous system.

These examples demonstrate the fact that severe trauma may have a local mechanical effect, a specific systemic effect, and a nonspecific effect on every cell of the body. The mechanisms involved in the causation of these effects appear to be physical (mechanical), neural, and humoral.

The Role of Wound Surgery

As long as nonviable tissue remains in a wound, the loss of blood and plasma continues; and the absorption of toxins of bacterial and tissue origin continues. Under these conditions, bacteria (including clostridia) may gain protection in the nonviable tissue outside the range of the defensive blood barrier. For these reasons, it is urgent that the nonviable tissue be excised as early as possible. The surgeon

excises the nonviable tissue and calls the procedure *débridement*. If he fails to *débride* the wound, nature does it for him. However, 2 weeks' time is required by nature; and the process is called sloughing. The rationale of *débridement* is: (1) to minimize the loss of blood and plasma, (2) to absorb bacteria or other toxins, and (3) to remove the dead tissue in order to effect proper circulation to the edges of the wound. In carrying out this procedure, only the nonviable tissue is excised. Some tissue may be injured but it is still viable, and it should not be excised.

The role of immediate surgery is to control hemorrhage, to determine the extent of injury, to excise nonviable tissue, to remove foreign bodies, to repair visceral, arterial, and other mechanical defects, and to prevent fluid collections by drainage.

The battle wound is a contaminated wound. Its bacterial flora usually includes clostridia, as well as a mixed aerobic flora. However, this may not be the basic reason for leaving wounds open after primary surgery. A contaminated wound can often be closed if it has been adequately *débrided*. In the patients studied in Korea (where the average evacuation time was 3.5 hours), personal experience included the *débridement* and primary closure of 20 soft-tissue wounds of muscle and an additional 20 soft-tissue wounds, including injuries to major arteries. Selection of wounds was based on the adequacy of *débridement*. They were small wounds, but contaminated. All 40 healed primarily. In their early management, wounds are left open for fear of inadequate *débridement* and resultant infection. There are several reasons why *débridement* may be inadequate. Sometimes adequate exposure may be almost unobtainable without creating serious deformities. After exposure, it may be quite difficult to delineate viable from nonviable tissue. Bleeding from tissue is not a reliable sign of visibility; nor is color of tissue a reliable index. The safest criterion is the contractility of muscle. Although excision of all noncontractile muscle is a safe index, it may require radical surgery, since the loss of contractility may be a reversible process. To date, although contractility is not completely satisfactory, it is perhaps the safest criterion.

As a generalization, it would appear that the better the natural blood supply to an area, the less tissue destruction there will be adjacent to a given missile trace and the more conservative the *débridement* required. For this reason, a functional compromise may be made and wounds of the scalp, face, thorax, stomach, small bowel, and blood vessels may be treated by a primary closure which results in normal healing in most instances.

Because of the fear of inadequate *débridement* and because of resultant dead space in the large wounds, primary *débridement* and delayed closure of soft-tissue wounds remain the soundest policy for most battle wounds. Under conditions in Korea during 1952 and

1953, where débridement was early, antibiotic therapy and tetanus prophylaxis were routine; and life-endangering infections were seldom encountered. The writer did not personally observe a case of tetanus. Gas gangrene and meningitis reached an all-time low in military medicine.

Section II. Shock and Transfusion

To appreciate shock following injury is to appreciate the entire field of trauma: the wounding agents, the nature of the injuries, the body's response to injury, the characteristics of the resuscitative agents, the effectiveness of the resuscitative methods, and the sequelae of injury, as all are entwined in the clinical syndromes found in the wounded soldier.

As more is learned about the nature of injury and the body's response to injury, the term shock will be discarded and in its place terms used will be the specific injury and the specific response. Such a step is almost justifiable now. The value of retaining such an all-inclusive term is only to focus attention on the serious deficiency of the circulatory system. There is no common cause of hypotension following injury and, therefore, no common therapy. A wound of the central nervous system may produce hypotension by injury to the sympathetic nervous system. An injury to the heart or pericardium may produce hypotension because of a primary deficiency in cardiac output. An open chest wound may produce hypotension presumably because of loss of the thoracic pump mechanism and result in a decrease in return of blood to the right heart and a diminution of cardiac output. These are specific wounds which cause specific deficiencies and require specific therapy. Classifying all such patients together in any single diagnosis or plan of therapy will result in added fatalities.

Hypotension and shock are therefore no more specific than fever or jaundice. Like the latter terms, however, they serve to focus attention on the gravity of the situation in the individual patient. During World War II, the Board for the Study of the Severely Wounded pointed out that hypotension may have many causes; but the chief cause in most battle casualties is a deficiency in blood volume.

This discussion of shock deals primarily with patients who have injuries of the abdomen and extremities. The present concept of shock, as developed in the Korean studies, is based on a knowledge of the nature of the injury and the body's response to the injury. The thoughts expressed here grew out of many informal conversations held at the litters of casualties at the 46th Surgical Hospital (Mobile Army) and in the laboratory of the Surgical Research Team of Korea. Many of the expressions are not original with this writer; and much of the work mentioned is the result of combined experiences.

When a patient has sustained a severe injury, there is a response on the part of every organ in the body in an attempt to compensate for and heal the injury. Like life itself, this compensatory mechanism is an interdependent mechanism; and it is dependent upon the circulation of blood from one capillary bed to another. The response is a continuing function. One element of the injury is blood loss. When blood loss is greater than 20 per cent, an inadequate circulation results. Hypotension is one manifestation. This state of circulatory insufficiency damages the various organs taking part in the compensatory effort. Circulatory insufficiency, which is produced by blood loss, therefore furthers the injury by destroying the defense mechanisms.

Shock may be defined as the clinical picture of an inadequate circulation following trauma. It is due initially to an inadequate volume of blood in circulation.

A massive wound, as stated previously, includes at least four elements of injury; and blood loss is only one of these elements. The others are tissue destruction, mechanical defects, and bacterial contamination. All of these elements of the wound produce a deleterious effect, frequently referred to as wound shock.

Tissue destruction produces a dynamic shock. It is not an injury which occurs for the moment only. Blood continues to be lost into the wound, while the toxins from bacteria and destroyed tissue are absorbed.

The entire body responds in order to meet the continuing injury. As a result of this response, compensation of the circulation may result. If the initial trauma is too great or if it is repeated and the resultant blood loss is excessive, circulatory insufficiency results. This does not imply a decompensation of the peripheral vascular mechanism. The autonomic system, heart, and peripheral vascular bed may be functioning maximally. If the injury and blood loss are of such magnitude that the compensatory mechanism cannot maintain an adequate circulation, hypotension results.

Most of the studies of shock have centered around the loss of blood; and herein lies the crux of the problem. Studies in the Korean conflict confirmed the earlier observations that hypotension results from a rapid loss of approximately 25 per cent of the blood volume. Up to this point, the heart and autonomic nervous system can compensate for the loss by increased cardiac rate and vasoconstriction in order to maintain a normal pressure. After the rapid loss of 25 per cent (about 1,200 cc.), hypotension develops and, if there is a rapid loss of up to 40 per cent (2,000 cc.), hypotension is profound.

Observations at the forward surgical hospital indicated that this hypotension could invariably be reversed before anesthesia, if the central nervous system was intact and if hemorrhage could be con-

trolled. There were no exceptions among nearly 5,000 casualties. Furthermore, in the resuscitation of these 5,000 battle casualties within an average time of 3.5 hours after injury, irreversible shock was not recognized prior to anesthesia provided hemorrhage had been controlled and provided no injury had occurred to the central nervous system. At this early stage, continued hypotension was the result of either continued hemorrhage or inadequate transfusion. Anesthesia, however, blocks part of the compensatory mechanism; and it may convert the compensated circulatory system to a state of profound shock. Moreover, shock may become extreme after anesthesia; and occasionally it may prove fatal if there is no response to repeated transfusions.

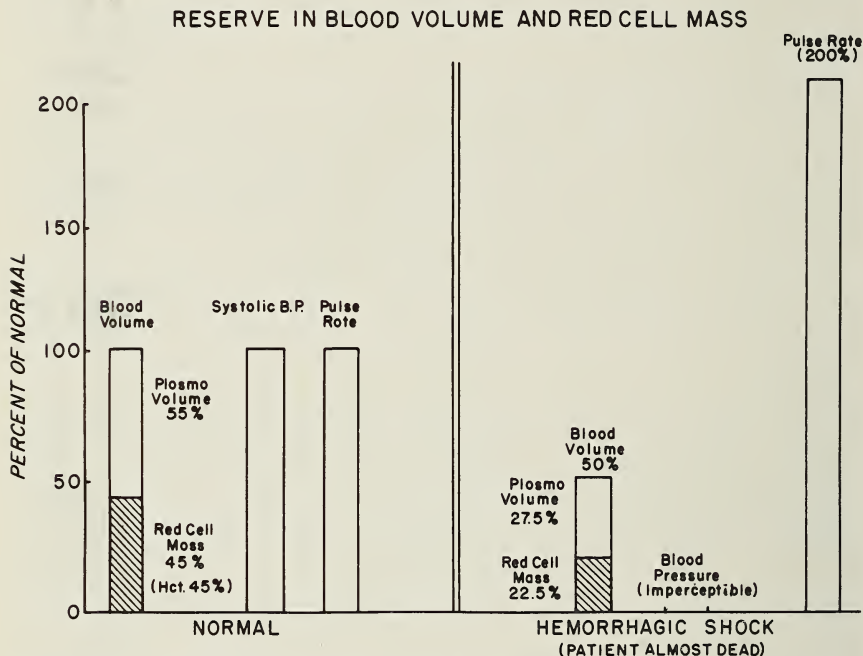


FIGURE 2. Hemorrhagic shock following the rapid loss of 50 per cent of the blood volume.

The purpose of the circulatory system is to carry substances from one capillary bed to another, for instance, from lungs to limb, from liver to brain, from bowel to liver or to heart. Circulatory failure is a failure of capillary circulation. This failure is brought on by a reduction in the circulating blood volume. If the volume is suddenly reduced 50 per cent, circulatory failure is profound and the blood pressure is unobtainable (Fig. 2). Under these conditions, total blood volume, plasma volume, and red-cell mass are each reduced by 50 per cent. Death is imminent. If the 2,500 cc. loss of blood is replaced by dextran in such an amount that the total blood volume is

restored, the red-cell mass remains at the low volume of 1,125 cc. and produces a lowering in hematocrit to 22.5 per cent. The red-cell mass, therefore, has not undergone any change. Upon restoration of blood volume, the patient will respond by a return of his blood pressure to normal; but his pulse rate will be somewhat fast, although slower than before therapy (Fig. 3). Proportionally, the reserve is far greater in red-cell mass than in blood volume. Circulating blood volume and capillary flow or pressure are the important elements. They are maintained by the use of plasma-volume expanders. These expanders proved to be satisfactory for use in the forward areas, where there was unavailability of whole blood.

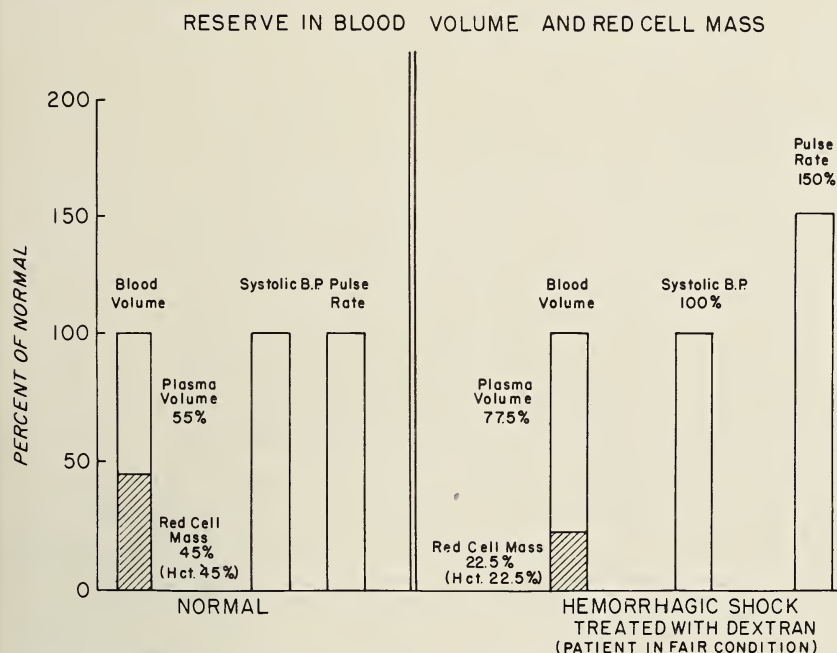


FIGURE 3. Schematic diagram of blood volume after dextran. Plasma volume is expanded by dextran and blood pressure rises. This shows the reserve in red cell mass which is greater, percentage-wise, than the reserve in blood volume.

The value of whole blood transfusions was recognized in World War II. In Korea, the helicopter permitted the treatment of many casualties who, under previous battle conditions, might have died in transit or would have been considered hopelessly injured. Blood given without crossmatching was literally poured into these wounded soldiers. This experience with massive transfusions was unprecedented. Often the question was raised as to whether or not an excessive amount of blood had been used. Clinical experience demonstrated that, if transfusion was slowed, hypotension and death resulted.

The summary of studies in blood volume following operation by Prentice and his associates unequivocally states the justification for massive transfusions in most of the severely injured. Both clinical experience and blood volume studies indicated that over transfusion rarely occurred.^{1 3}

Artz³ summarized the studies of 38 consecutive patients whose blood pressure at the time of admission to the hospital was zero as measured clinically. During the first 24 hours, 12 of the patients required less than 15 pints of blood. No fatality occurred. There were 26 patients who required more than 15 pints of blood; and the case fatality rate was 54 percent. This study demonstrates that the amount of blood required for resuscitation is a better index of prognosis than is the blood pressure at the time of admission. In the group requiring more than 15 pints, the case fatality rate among the 10 patients with wounds limited to the abdomen was 90 percent in contradistinction to a case fatality rate of only 22 percent in the nine casualties with wounds of the extremities. This latter comparison indicates the difficulty in controlling intra-abdominal hemorrhage. More immediate and beneficial results will be obtained from further studies on methods of controlling hemorrhage rather than from studies on the mechanisms of late shock.

In the same summary, 84 casualties (irrespective of blood pressure on admission) required 15 or more pints of blood in the first 24 hours. Continued hemorrhage was again recognized as a major factor accounting for 16 deaths. The data of Prentice and his colleagues indicated that many of the others died of deficiency in blood volume, despite massive transfusion and apparent hemostasis.¹ Furthermore, a sharp difference was noted between the mortality in the patients with abdominal wounds and those with extremity wounds.

The final statement of these investigators was that, in this group requiring massive transfusion, posttraumatic renal insufficiency developed most frequently. This complication is a direct or indirect result of the magnitude of the wound and the severity of the shock. Posttraumatic renal insufficiency cannot be predicted by evaluating the magnitude of the wound or the severity of the hypotension at the time of admission. It can best be predicted by response to transfusion. A hypotensive, seriously wounded patient whose blood pressure responds sluggishly to transfusion, in the face of apparent hemostasis, is a likely candidate for renal failure.

The primary cause of hypotension is a deficiency in blood volume. A deficiency of the sympathetic nervous system could seldom be demonstrated. At present, vasoconstrictors would appear to have only a limited place in the treatment of shock in a young soldier who becomes a battle casualty. Their place is limited to meeting a deficiency in the

function of the autonomic nervous system, a deficiency which was recognized only when the system was blocked by anesthesia or when there was an anatomic wound of the central nervous system. Repeated studies were made of the role of vasoconstrictors in the treatment of the casualty with postoperative refractory shock. The blood pressure could be maintained for a few hours; but death was almost inevitable.

Clinical experience and blood-volume measurements demonstrated the value of massive transfusions. In spite of continued transfusion and apparent hemostasis, some casualties died following anesthesia and operation from refractory shock. In a few of these patients, pulmonary edema was demonstrated at autopsy. Although the cause of hypotension initially was blood loss, it appeared evident that cardiac failure might develop following massive transfusion. Secondary shock was not infrequent following anesthesia and operation. This type of shock usually responded to additional transfusion, as it represented blood-volume deficiency and possibly a continued diminution in the function of the sympathetic nervous system due to the anesthesia. The occasional patient who did not respond postoperatively to blood transfusion died with pulmonary edema and, presumably, cardiac failure. Studies of the seriously injured patients on the day after operation often revealed a transient rise in the potassium concentration of the plasma; and an occasional patient showed a dramatic response to an infusion of calcium gluconate.

Circulatory failure means the failure of circulation within the vascular tree. Of greater interest is the circulation, or "diffusion," between cells and extracellular fluid and between extracellular fluid and blood. This extravascular circulation, or diffusion, is the factor which determines cellular function and life. Capillary circulation is the only means of providing it. A rough approximation of this total body circulation, or mixing, can be gained from experiments with deuterium oxide. When deuterium oxide is given intravenously to a normal subject, diffusion from the blood is immediate and equilibration occurs quite rapidly. In our studies of total-body diffusion and mixing, the mixing was slightly slower; but, when deuterium oxide was given intravenously, the resultant curve—the deuterium concentration in venous blood of the opposite arm—revealed the rapid mixing and diffusion. A similar study of a patient in shock demonstrated a greatly retarded mixing. This could scarcely be due to a slower circulation time. It was because of a decrease in the effective capillary circulation, *i. e.*, in the extracellular and intracellular diffusion.

This decrease in the effectiveness of the total-body circulation leads to an exaggeration or deviation of the response of the function of every organ. If shock did not persist too long, no dangerous failure in organ function was found. The blood volume was decreased and the total circulation was depressed. The hematocrit fell following wounds of the extremity and rose after abdominal wounds. The

plasma sodium fell and sometimes the plasma potassium rose. The function of the autonomic nervous system appeared clinically intact, as evidenced by clinical studies and by the functional studies by Stahl.² The adrenal cortical response developed rapidly, in spite of severe shock, as indicated by: (1) the fall in eosinophile counts, (2) the urinary retention of sodium and water, (3) the diuresis of potassium, and (4) the increased excretion of corticosteroids. Hepatic function, as measured by standard liver function tests, was depressed; but as measured by the more vital tests of metabolism it appeared generally adequate. Renal function appeared in some aspects to be markedly depressed, presumably due to an exaggerated renal vasoconstriction. The clotting mechanism, the hematologic response, the bacterial defense, all appeared adequate in the face of shock of short duration.

If shock continues for a long period of time, cellular damage becomes so severe that the cells, organs, or systems may lose their function. The heart or brain may be the first vital organ to deteriorate. Shorr felt that the liver—by release of ferritin (a vasodilator)—makes prolonged shock refractory. Studies in Korea indicated that many of the casualties who had been resuscitated from critical injuries maintained a positive V. D. M. test (ferritinemia) for several days. Fine suggests that the release of bacteria or bacterial toxins from the bowel makes prolonged shock refractory. Bacteremia was seldom demonstrable in the battle casualty and the results of cultures of peripheral blood could not be correlated with the degree or duration of shock or the ultimate fate of the casualty. In those most seriously wounded casualties in Korea who survived, renal damage appeared to be the most deleterious residual damage.

Summary

The wound is a dynamic injury—not a static one—as it continues to insult the body. The insult is greatly lessened by débridement. The initial treatment of wound shock is to restore and maintain blood volume, preferably with blood, followed by surgical correction of the wound. The body responds in its entirety to severe trauma. By blocking this response, anesthesia is an additional injury. Although anesthesia is necessary in order to correct surgically the deleterious effects of the wound, actually it furthers the injury.

At an average time of 3.5 hours after injury, irreversible shock was not recognized prior to anesthesia in several thousand casualties of the Korean conflict. Following anesthesia and operation, hypotension was occasionally quite refractory, but usually responded to continued transfusion. Even after the use of massive transfusions, the blood volume was surprisingly low.

It appears that, following injury, the function of every organ and system in the body is altered. The alteration is proportional to the magnitude of the original injury in both magnitude and duration. Hypotension accentuates the changes.

Because the wound produces a continuous deleterious effect, resuscitation must be continuous until the response of the body can compensate for the effect of the injury.

Section III. Clinical Experiences with Wounds of Various Anatomic Areas

The basic defects after injury center around two points: the loss of blood and the local wound. The syndromes of injury therefore vary as the blood-volume deficit varies and as the local lesion affects various functions.

Although the systemic effects of a wound are primarily due to its loss of blood, there are other specific effects of the wound. The purpose of this report is to summarize the clinical observations in the resuscitation of almost 5,000 battle casualties at the 46th Surgical Hospital (Mobile Army) in Korea.

Cranial Injuries

The syndrome following wounds of the brain is dominated by the neurologic picture. The state of consciousness is depressed to a varying extent, depending upon the exact anatomic area of the wound. Blood loss is seldom a predominant feature of the injury. The pulse rate is usually slow (50–80 per minute), being about 100 in only 10 percent of the patients studied. The blood pressure varies tremendously from patient to patient. A slight hypertension of 140/80 is not infrequent, nor is a hypotensive level of 80/40.

When the systolic blood pressure was correlated preoperatively with the blood-volume deficit (Evans blue-dye method), a qualitative relationship was found. The blood loss was not of great proportion and did not exceed 30 percent in the 11 patients so studied. The peculiarity of these patients was that hypotension might follow a loss of only 10 to 15 percent of their blood volume. Furthermore, it was observed that the blood pressure of such a casualty with a moderate hypotension often responded readily to a transfusion of only 500 cc. of blood.

Severe hemorrhagic shock was almost never seen following penetrating cranial wounds. Although blood loss is a factor, the predominant defect lies not in blood loss but in loss of neural function, often including the response of the autonomic nervous system.

The therapeutic implications are obvious. Blood loss should be replaced and further brain damage should be prevented by control of bleeding and débridement. At a forward echelon, infection did not appear to be a major problem.

Spinal Cord Injuries

Experience with these injuries was limited. The syndrome is one of hypotension and of paralysis below the site of injury. In the injuries of the lower cord, the sensorium may be perfectly clear. One instance occurred in which the patient was lying flat, reading a newspaper. His blood pressure in the arm was unobtainable and he was anuric. He died a few hours later, but his sensorium remained clear until shortly before death. His pulse was scarcely palpable at any time, a feature which could not be satisfactorily explained although the observation was confirmed in two subsequent patients.

The primary defect is not blood loss; and fatal pulmonary edema resulted when this fact was not recognized until overtransfusion had occurred. The primary defect is loss of function of the autonomic nervous system and possibly loss of proprioceptive reflexes to the cardiovascular system. Therapy is unsatisfactory, consisting at present of replacement of blood loss, support with vasoconstrictors, and operative débridement and decompression.

Thoracic Injuries

Severe chest injuries were rather infrequent at the hospital, partly because of the protection offered by the armored vest and partly because of immediate fatalities from the more serious injuries.

Two defects occur in a penetrating chest wound. One is blood loss; the second is an open thorax. The low pressure in the pulmonary system seldom permits rapid bleeding from the lung, although bleeding from intercostal arteries may be quite brisk. Blood loss may reach serious proportions as bleeding continues. However, hypotension is due, in part, to the open thorax. Closure of the open wound restores the effectiveness of the thoracic pump mechanism; hence it is an integral factor in correcting the basic defects. Then thoracotomy becomes necessary in this group of patients. It should be recognized that the open thorax and the lateral position of the patient contribute to the resulting hypotension. Failure to take cognizance of this fact in three casualties and to attribute the hypotension to continued blood-volume deficit led to overtransfusion to a serious extent.

Arterial Wounds

These are wounds in which hemorrhage is the primary injury. Tissue damage may be minimal and the responsiveness of the patient appears intact. As a result, the casualty often exhibits a marked vasoconstriction and tachycardia. By these mechanisms his systolic pressure is maintained at a near-normal level until a critical blood deficit is reached, after which the pressure falls rapidly. If the hypotension is not maintained for a prolonged time, replacement of blood volume coincides with a prompt restoration of a normal blood pressure. Therapy of this patient is not a problem. Because of the normal

blood pressure, the blood-volume deficit may not be appreciated prior to anesthesia and a severe hypotension may follow induction. Because of the frequency of this error in judgment, the surgeons developed the maxim: "Give the patient with an arterial injury a liter [of blood] more than he appears to need."

Traumatic Amputation

This is a far different wound from the simple, arterial wound. It results from the transmission of a tremendous amount of energy from an exploding mine or shell fragment. The tissue injury extends throughout the extremity if not throughout the body. There is local hemorrhage, bone, and muscle destruction. The relationship of muscle destruction and hemorrhage in producing the syndrome is not yet clear, but many of these patients respond very slowly to transfusion.

A bilateral, traumatic amputation of the thigh is the most massive injury seen in battle. The blood pressure on admission is usually imperceptible and the response to transfusion is very sluggish. This patient's defect is not explained on the basis of external blood loss only; and sometimes it cannot be explained. This patient represents shock in its most severe form—hypotension from blood loss and tissue injury, with possible impairment of the compensatory mechanism.

Abdominal Injuries

Many of the major therapeutic problems lie in this group of casualties. The extent of injury varies tremendously. Perforations of the spleen and stomach offer few problems. Perforations of the liver, kidney, small intestine (especially duodenum), or colon increase the gravity of the injury. Perforation of the inferior vena cava or the iliac, renal, or other major vessels, leads to critical problems in restoration and maintenance of blood volume. Hemorrhage may be so rapid that the blood-volume deficit cannot be corrected. The induction of anesthesia to control hemorrhage in such a patient may prove fatal.

The initial syndrome in the more severe injuries is that of the typical shock picture of hypotension and tachycardia. The hypotension is sometimes resistant to transfusion. These patients, having abdominal wounds complicated by severe hemorrhage, constitute the majority of fatalities. Progress will result by finding means of controlling hemorrhage and replacing blood-volume deficit prior to the added insult of general or spinal anesthesia. One obvious clinical lesson had to be re-learned; it was not to transfuse into the veins of the lower extremities when injury to the inferior vena cava was a possibility.

The peculiarities of abdominal injuries, which are shared in several aspects with the most profound of the extremity wounds, are the resistant hypotension, the high incidence of postoperative hypotension, the relatively high incidence of posttraumatic anuria, paralytic ileus, and hemoconcentration. The basic factors underlying these phenomena are not understood.

Retroperitoneal Trauma

This injury is characterized by profound shock and it was seen most frequently in crushing nonpenetrating injuries. Like the occasional patient with acute pancreatitis, the hypotension seems out of proportion to the blood loss and responds slowly to transfusion.

Fracture of Long Bones

This injury, like the traumatic amputation of the foot at the ankle, is one of the few wounds associated with severe pain. The peculiarity of the vascular response is the high incidence of hypertension. At the time of admission, many of the patients have a systolic pressure above 150 and sometimes as high as 240 mm. mercury. The pulse may be rapid; more frequently, however, it is between 60 and 80. The skin is warm; and the pulse volume is fairly good. Relief of pain may be obtained and the patient may fall asleep. Nevertheless, hypertension usually persists. It is dropped sharply by blockade of the autonomic ganglia with hexamethonium. The hypertension is undoubtedly mediated through the autonomic nervous system. Although these patients frequently demonstrated a precipitous drop in pressure following pentothal induction, their subsequent course was smooth.

Nonviable Tissue

In the presence of an ischemic, nonviable limb, shock may be somewhat refractory to transfusion. To date, an occasional, unexpected death within the first 24 to 48 hours after an injury can be explained only on the basis of toxicity or continued insult from such a wound.

Each organ or system serves an integral function in the body's economy. To injure a specific system is to disrupt at least one function of the body. It is, therefore, not surprising that wounds of various anatomic areas produce different defects and present different clinical manifestations.

Recognition of the clinical manifestations of injury and an understanding of the underlying physiologic alterations are not only essential to good clinical surgery, but they are essential to further progress.

References

1. Prentice, T. C., Olney, J. M., Jr., Artz, C. P., and Howard, J. M.: Studies of Blood Volume and Transfusion Therapy in the Korean Battle Casualty. *Surg., Gynec. & Obst.* 99: 542, 1954 (Chapter 9 in Volume II of this series).
2. Stahl, R. R., Artz, C. P., Howard, M. M., and Simeone, F. A.: Studies of the Response of the Autonomic Nervous System Following Combat Injury. *Surg., Gynec. & Obst.* 99: 595, 1954 (Chapter 6 in Volume I of this series).
3. Artz, C. P., Howard, J. M., Sako, Y., Bronwell, A. W., and Prentice, T.: Clinical Experiences in the Early Management of the Most Severely Injured Battle Casualties. *Ann. Surg.* 141: 285, 1955 (Chapter 2 of this volume).

Part Two

Management
of Specific Clinical Problems

Chapter 8

Acute Vascular Injuries in the Korean Conflict:

An Analysis of 77 Consecutive Cases*

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The sudden disruption of major arterial continuity, so frequently seen in battle casualties, confronts the military surgeon with a serious and discouraging problem. If death from massive hemorrhage does not occur, a high percentage of these patients rapidly pursue the inevitable course of ischemia and gangrene, necessitating early amputation of the whole or part of an extremity. In the interval between every war, outstanding discoveries have been made in most fields of medical research. The encouraging results of these have been the ultimate reduction in mortality of the wounded reaching a forward medical installation to less than 2 per cent. During the past 10 years, many experimental and clinical contributions have been made in the realm of cardiovascular surgery which have provided satisfactory solutions to problems previously unsolved and opened unlimited horizons for further research. Despite these advances, there has been no significant decline in the number of limbs lost following major vascular injury in battle casualties. This, undoubtedly, can be attributed to the fact that the recommended treatment in the majority of cases, namely, immediate ligation of the bleeding artery, has remained unchanged since World War I or even earlier.

Prior to the numerous surveys made during World War II, there were no really accurate statistics regarding the incidence of gangrene and subsequent amputation following ligation of a damaged artery. Makins' monumental work on vascular injuries in World War I showed an over-all amputation rate of 18.1 per cent and in popliteal injuries, an amputation rate of only 43 per cent.¹⁴ Others have reported up to 70 per cent gangrene following early ligation of major vessels and up to 100 per cent in ligation of the popliteal artery. ^{1 4 16 17 24 25}

De Bakey and Simeone ⁴ have partially explained this inconsistency

*Previously published in *Annals of Surgery* 138: 158, 1953.

in figures by showing that previously reported series contained many cases of false aneurysms and even simple vein ligations. Neither of these lesions was in any way comparable to acute arterial injury and ligation. In addition, some vascular lesions were not basically counted as such because of associated infection and/or massive trauma.

It was not long after the onset of World War II that it became clear that the sequelae of early arterial ligation were more serious than had been reported. The statistics of De Bakey and Simeone,⁴ Rose, Hess, Welch,¹⁷ and others^{1 16 24 25} showed an incidence of gangrene varying from 45 to 55 per cent when a major artery was initially ligated. It was realized that some method of approach other than ligation would have to be utilized if more limbs were to be saved. In selected cases, those with small wounds or small areas of arterial damage, repairs were attempted. The methods used were, for the most part, the direct suture repair, the nonsuture vein graft, and the nonsuture tube anastomosis. The over-all results were disappointing in that the incidence of gangrene, even in these selected cases, varied between 36 and 50 per cent. This was questionable improvement over the results of immediate ligation, especially since the reparative treatment was time-consuming and required a great deal of specialized technical skill. De Bakey and Simeone⁴ found that the incidence of gangrene following the nonsuture tube anastomosis was 50 per cent and following the nonsuture vein graft was 57 per cent, even higher than the 49 per cent incidence after ligation. There was only slight improvement following suture repairs, with an amputation rate of 35.8 per cent. The adjunctive use of sympathetic block and fasciotomy was of questionable value.

In view of the statistical evidence accurately recorded from World War II, the policy at the beginning of the Korean conflict was to ligate all vascular injuries unless they were of such size that transverse suture repair or simple end-to-end anastomosis was possible. The destructive force of the modern military weapons made the number of lesions suitable for repair quite small. Reports from the Far East^{13 23 26} have indicated that the incidence of success, even in these cases, was quite low. Some series have been reported in which over 70 per cent of the repairs failed.

It has been observed that in some instances vessels have been repaired only to develop a thrombosis later. Some of these patients have required amputation of the involved extremity at a higher level than would have been anticipated initially. This, it was believed, was due to propagation of the thrombus up and down the vessel with occlusion of important collateral channels. However, this was only an impression based on personal communications with numerous surgeons in forward areas and was not confirmed by statistical proof.

Scattered reports on several small series of cases in which grafts were inserted in arterial defects are also available from the Korean conflict.^{13 26} The circumstances concerning their use in regard to the extent of the injury, the time-lag from wounding to repair, associated fractures, and other factors (to be discussed later), which may be of greater or lesser importance in the ultimate outcome, are not available. The results, though, have been most discouraging. The incidence of failure in one series of six cases was 100 per cent.¹³

In contrast to the universally poor results obtained in repair of early vascular lesions were the results reported from this hospital in the repair of late vascular lesions.¹⁹⁻²¹ The series consisted of 150 arteriovenous fistulas and false aneurysms in Korean war wounded. Ligation and excision were performed in the first 26 cases involving major arteries. The results were similar to those of Elkin^{6 7} and others.²² There were no cases of gangrene requiring amputation. It is significant, however, that 30 per cent of the patients presented definite symptoms of arterial insufficiency, even with the adjunctive use of sympathectomy. Reparative surgery in 67 cases of major arterial lesions by other methods such as the end-to-end suture anastomosis, the homologous arterial graft, and the autogenous vein graft gave much better functional results. Thrombosis occurred in only two patients. The over-all results were so satisfying that it was established that all major arterial lesions would be repaired by anastomosis or graft.

It is not meant to imply that one can in any way compare the results of repair of acute arterial injuries with repair of arteriovenous fistulas and false aneurysms. In the former group, many factors enter which are never encountered in the latter group. Collateral circulation has had a chance to develop in the chronic lesions so that even ligation is followed by an extremely small incidence of gangrene. Russell¹⁸ has shown that the collateral circulation can develop sufficiently in a few weeks, and in many cases within a few days, to significantly lower the incidence of gangrene following arterial ligation. The important point to be emphasized, however, is that it was believed that certain technics perfected during the study and treatment of the fistulas and aneurysms could be applied to the acute vascular injuries and significantly reduce the incidence of gangrene.

This manuscript reports the initial results and follow-up obtained in a series of 77 consecutive vascular injuries treated by one of us (E. J. J.), a member of the Surgical Research Unit from the Army Medical Service Graduate School.

Material

All patients in this series were treated during a 4-month period in Korea at the Mobile Army Surgical Hospital, 8055th Army Unit. Approximately 75 per cent were members of the Eighth United States Army. The remaining 25 per cent was divided among soldiers of the British, Canadian and Republic of Korea Armies, and Chinese prisoners of war.

The vascular injuries were divided into two main groups: those with injuries to minor arteries and those with injuries to major arteries. There were 43 patients, or 55.8 per cent in the former group, and 34 patients, or 44.2 per cent, in the latter group. Arteries classified as minor were the brachial profunda, radial, ulnar, femoral profunda, anterior tibial, posterior tibial, peroneal, or any combination of these vessels. Major arteries were considered the carotid, axillary, brachial, iliac, femoral and popliteal (Table 1).

Table 1. Distribution of 77 Consecutive Vascular Injuries Treated and Incidence of Gangrene

Vessel	Number of Cases	Per Cent	Loss of Limb	
			Number of Cases	Per Cent
Common carotid	1	1.3	-----	-----
External carotid	1	1.3	-----	-----
Axillary	5	6.6	-----	-----
Brachial	12	15.7	-----	-----
above profunda	4	5.2	-----	-----
below profunda	7	9.0	-----	-----
profunda	1	1.3	-----	-----
Radial	6	7.9	-----	-----
Ulnar	5	6.6	-----	-----
Radial and ulnar	2	2.6	-----	-----
External iliac	1	1.3	-----	-----
Femoral	14	18.5	2	14.2
above profunda	1	1.3	-----	-----
below profunda	8	10.6	1	12.5
profunda	5	6.6	1	20.0
Popliteal	7	9.0	2	28.5
Anterior tibial	6	7.9	1	16.6
Posterior tibial	9	11.4	3	33.3
Both tibials	2	2.6	-----	-----
Peroneal	3	4.0	-----	-----
Anterior tibial & peroneal	1	1.3	-----	-----
Posterior tibial & peroneal	1	1.3	-----	-----
Both tibials & peroneal	1	1.3	-----	-----
Total	77	100	8	
Average				10.6

Factors Affecting Treatment

In all types of surgery the ultimate results are dependent upon, or are at least partially influenced by, many factors. The interplay of these factors is often clear-cut and well understood. Vast amounts of well-documented evidence have firmly established the criteria as to operability and probable outcome. Such is not the case with the acute vascular injuries. These lesions, though quite uncommon in general civilian practice, constitute approximately 1 per cent or more of all battle casualties. Therefore, data of statistical significance can best be obtained during periods of armed conflict. The exigencies of the military medical service, from a tactical standpoint, often preclude the compiling of complete and accurate medical records. Despite this unavoidable handicap, surveys from World War II have enumerated and explained many factors common to vascular injuries which are instrumental in determining the final results of treatment. This series was studied on the basis of these findings. Several other observations were made which were felt to be significant in the handling of the acute arterial interruption.

Type and Size of Wound

The day of the sword, spear and cannon ball as major instruments of warfare has long passed. Modern military weapons, with their high muzzle velocity and their tremendous propensity to fragmentation, aimed at inflicting mass casualties, present an almost incalculable destructive force. Rarely is the military surgeon fortunate enough to see a clean-cut knife wound or a small round bullet wound associated with a vascular injury which is usually a simple transection of the vessel or a small laceration in its wall. Such a lesion can often be adequately and successfully repaired by direct suture anastomosis or transverse suture of the laceration. Even if the repair fails, the collateral circulation is undisturbed and will usually preserve the viability of the limb. In most cases, however, the military surgeon is confronted with a large, jagged and gaping wound. This may be single, but in a high percentage of cases it will be multiple. The damaged artery will be destroyed for large segments and a simple repair will be impossible. In addition, it has been previously shown that the larger the wound, the greater the chance that the vital collateral circulation will be destroyed. It may also be implied that the effectiveness of the collaterals will be proportionately decreased with the multiplicity of wounds in the extremity. Further accessory channels may be damaged with the performance of a complete and adequate débridement. On this basis, it would seem almost mandatory that some type of reparative surgery be instituted to re-establish the major channel, especially in such vital areas as the axillary, iliac, common femoral and popliteal.

Studies on wound ballistics have shown that the damage inflicted by modern military weapons is not confined to the actual path of the missile. At the time of wounding, a large temporary cavity, many times the size of the actual injury, is formed in the tissues. This is not confined by the fascial planes. In this manner, arterial wall damage may occur far beyond that observed by the naked eye. This damage may not be sufficient to cause thrombosis in the areas where injury has resulted from cavitation. It is not inconceivable, however, that thrombosis may occur in arterial injuries associated with multiple wounds of the extremity, where numerous destructive cavities may be formed. In some areas, these cavities may overlap and the cumulative damage on the arterial wall may be sufficient to serve as a nidus for thrombus formation. In this type, failure of a vascular repair is not due to technical factors, but is based on a condition far beyond the control of the surgeon.

Of the 34 major vascular injuries in this series, it was found that only four (11.9 per cent) were caused by small-arms fire. Artillery, mortar, mine and hand-grenade fragments accounted for the remaining 30 (88.1 per cent). As would be expected from this, over 60 per cent of the patients had more than one wound. The amount of gross soft-tissue loss in the wounds associated with the major vascular lesions was compared. Small wounds were found in six, or 17.7 per cent of the cases. Medium-sized wounds were observed in 17 (50 per cent), and massive wounds in 11 (32.3 per cent). The condition of the artery observed at operation closely paralleled the magnitude of the wound and will be discussed later.

Location of the Wound

Results of detailed statistics from World War II have shown that great prognostic value could be placed upon the location of an acute vascular injury. De Bakey and Simeone⁴ reported the incidence in the lower extremity to be almost twice that of the upper extremity, with 1,561 cases in the former and 892 cases in the latter. Of these cases, 774 (49.6 per cent) in the lower extremity resulted in amputation, while only 214 (24 per cent) in the upper extremity went on to amputation. It was also observed that in the brachial and femoral artery, considerably different results were obtained, depending on whether the lesion was above or below the profunda branch. The incidence of amputation following brachial artery injury below the profunda was 25.8 per cent. This rose to 55.7 per cent when the lesion was above the profunda. Almost as dramatic results were noted in the femoral artery with an amputation incidence of 54.8 per cent in lesions below the profunda and 81.1 per cent in lesions above this branch. When it is considered that the majority of all these vascular

lesions were ligated, it is obvious that the collateral circulation must be quite inadequate in areas with the greatest percentage of amputation. Here again one must consider vascular repair, especially in the critical areas, as essential for limb preservation.

It was found that of the 77 vascular injuries reported, 32 (41.5 per cent) occurred in the upper extremity, and 45 (58.5 per cent) occurred in the lower extremity (Fig. 1). Amputation following any of the upper extremity lesions was not necessary, but was performed in eight (17.7 per cent) of the patients with lesions located in the lower extremity. When tabulating the lesions of the major vessels of the lower extremity, three cases (17.6 per cent) came to amputation as compared with 62.1 per cent reported by De Bakey and Simeone.⁴

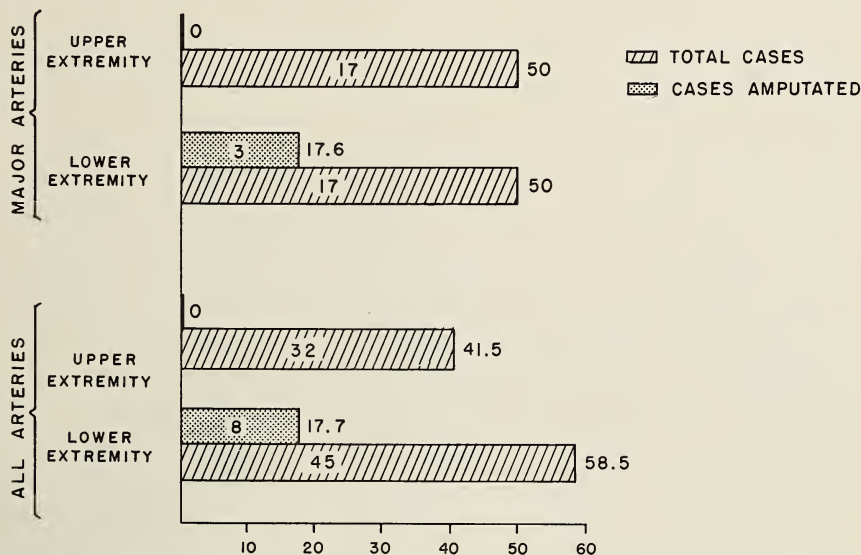


FIGURE 1. Incidence of amputation compared with location of vascular injury.

Again in our series there were no amputations following lesions either above or below the brachial profunda artery. In the femoral area, the only amputation performed followed a lesion below the profunda. This represents an incidence of amputation of 12.5 per cent.

It is also of interest that lesions involving so-called minor vessels resulted in amputation in five cases (11.6 per cent) of the 43 cases in that group. The highest percentage followed lesions of the posterior tibial artery, there being three (33.3 per cent).

These figures seem to indicate that with the routine utilization of vascular repair in all major arterial injuries, the incidence of amputation can be significantly reduced. In this series, that incidence is even less than in the minor vessels which were ligated. This is

especially significant when it is realized that the entire group of cases were uncontrolled and unselected.

Associated Injuries

It has been mentioned previously that over 60 per cent of this series had other wounds in addition to vascular disruption. In the majority of cases, however, these associated wounds were noncritical (not acutely life-endangering) and their débridement was delayed until the vascular repair was completed. In a few cases where associated abdominal or chest wounds were present, the vascular repair was still given preference. The average time necessary for repair varied from 15 to 45 minutes, depending upon whether an anastomosis or a graft was necessary. No case was observed in which it was felt that the order of preference adversely affected the ultimate recovery of the patient. The only patients who required immediate surgery, without the benefit of adequate preoperative resuscitation, were those few with uncontrollable hemorrhage and persistent shock, despite massive whole blood replacement by the intra-arterial route. Even in these, the indication for immediate surgery was vascular damage. Certainly all patients in whom treatment could be delayed for resuscitation from 1 to 3 hours or more could well afford an additional delay of 30 minutes to permit a vascular repair, and thus perhaps save a limb. More and more, the opinion has been expressed that all surgery should be delayed until adequate resuscitation has been performed and the patient's apprehension concerning his wound and impending surgery allayed. This would give additional support to the premise that vascular repair should be given early priority.

Of the major vascular injuries seen, shock was present to a greater or lesser degree in 52 per cent. Of this group, 16 per cent were in profound vascular collapse and were treated by intra-arterial transfusion. Much credit must be given in this regard to the Battalion Surgeon for his initial resuscitation, many times with whole blood or dextran. Several of the patients with major vascular injury were entirely resuscitated with dextran. In these cases, the erythrocyte count a week after surgery was still less than two million cells per cu. mm. and the hematocrit was in the range of 15 to 20 per cent. In no case did this prevent successful vascular repair or result in loss of a limb, despite the obviously prolonged period of decreased oxygen-carrying power of the circulating blood.

The incidence of fractures and nerve injuries associated with the vascular lesions was also studied. Often a patient arrived at the Mobile Army Surgical Hospital suffering from exposure and profound shock. Both legs and arms were cold and pulseless. It was almost impossible to determine accurately whether a vascular injury was present or whether the condition of the extremity represented a combination of exposure, shock, and/or vascular spasm. In many of

these cases, the decision to explore the limb for a vascular injury was at least partially based on the presence or absence of associated fractures and nerve damage.

It was found that in patients with axillary artery injury, associated fractures were present in only 20 per cent as compared with 80 per cent with nerve damage. In 20 percent there were combined fractures and nerve injury. This might well be expected when it is recalled that the axillary artery is surrounded by the cords of the *brachial plexus*. Bone and nerve damage, in association with brachial artery injuries, were quite common. The percentage was 63.6 and 81.8 respectively. Combined damage was found in 54.5 per cent. This also might be anticipated, since the artery is not only accompanied by the

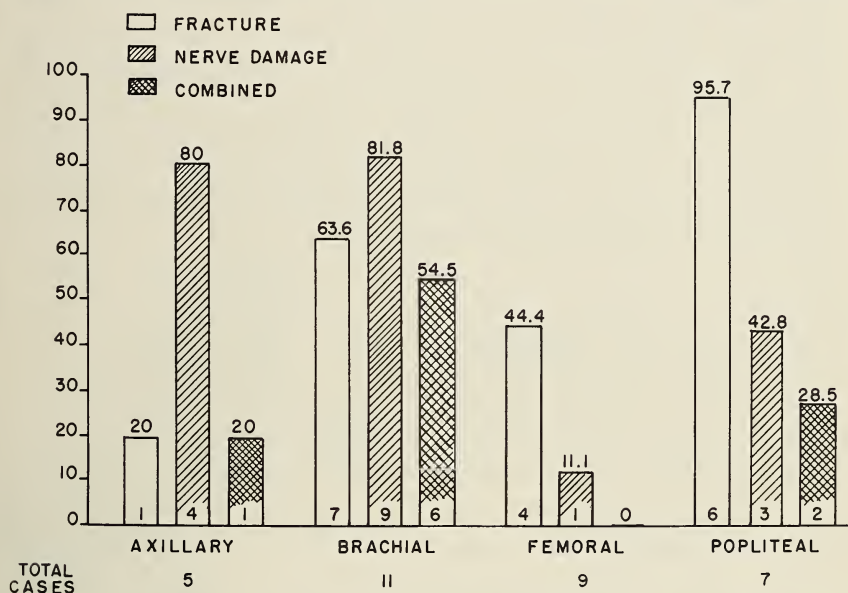


FIGURE 2. Incidence of associated fractures and nerve damage in major extremity arterial injuries.

median and ulnar nerves, but lies in close proximity to the humerus. Of the patients with femoral artery lesions, 44.4 per cent had fractures in the involved limb, while only 11.1 percent had neuropathy, since the vessel lies close to the femur but quite a distance from the sciatic nerve. Associated fractures were very common in patients with popliteal artery injuries. They constituted 95.7 per cent of the cases as against only 42.8 per cent with associated nerve damage. Combined lesions of the bone and nerve were found in 28.5 per cent (Fig. 2).

It cannot be inferred from these figures that any definite percentage of patients with fractures and nerve damage may have arterial injuries. On the other hand, the presence of bone or nerve injury in

certain areas implies a sufficiently high percentage of vascular injury to justify giving surgery a priority, which would lessen the incidence of amputation.

Condition of the Artery

Care was taken in all cases of major vascular damage to record the condition of the artery observed at the time of the operation. In contrast to the results reported by De Bakey and Simeone,⁴ no difference was found in the incidence of amputation following arterial repair whether the artery was initially severed, lacerated, or had undergone thrombosis. It did make a difference in the type of repair utilized, but not in the final results. The lacerated vessels varied from those with small tears in otherwise intact walls, to those with segmental defects of two inches held together by only strands of adventitia. Whenever any tissue of the arterial wall connected the ends of the vessel, it was considered a laceration. Cleanly severed arteries were noted in only two cases. All others had jagged defects varying from 1 to 5 inches in length. Thrombosis of an intact artery was found at the time of the operation in only one case. This was a patient with a large popliteal wound.

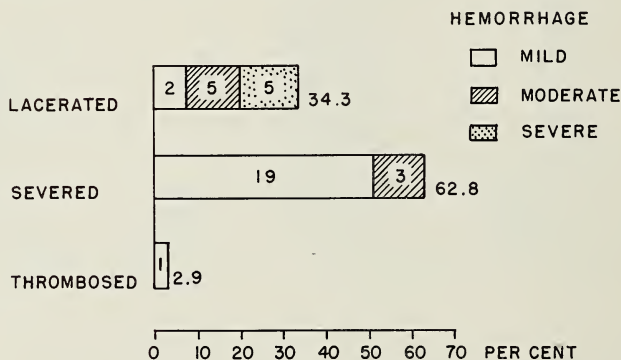


FIGURE 3. Condition of vessel compared with hemorrhage in extremity in arteries repaired.

A study was also made of the condition of the artery at the time of operation as compared with the amount of hemorrhage in the surrounding tissue. As expected, the severed arteries retract into the surrounding muscle, then contracted and underwent thrombosis. The lacerated arteries could not retract into the tissue and the formation of large hematomas resulted. Lacerated arteries were found in 12 cases (34.3 per cent). In one case (2.9 per cent) the vessel was intact but thrombosed. Of the patients with lacerated vessels, 83.3 per cent had moderate to severe hemorrhage in the extremity. In 16.7 per cent the hemorrhage was mild. In patients with severed vessels where

retraction was possible, 86.3 per cent showed minimal hemorrhage in the extremity, and only 13.7 per cent had moderate to severe hemorrhage (Fig. 3).

The degree of hemorrhage in the extremity was also of value in helping to determine which patients had vascular injuries. A large tense hematoma in the limb almost always indicated a vascular injury, usually of a lacerating nature. Arteries which were severed or thrombosed could not be diagnosed by the extent of hemorrhage in the surrounding tissue.

Time-Lag

Probably one of the important, if not the most important, factors which predetermines the final outcome of vascular injuries is that described as time-lag. This term refers to the time elapsing between the moment of injury and the initiation of surgical treatment. The upper limit which had been used previously was arbitrarily set at 6 to 8 hours. It was felt that treatment of arterial lesions beyond that time would rarely be successful. Thromboses which probably have occurred in the distal arteries, and changes in the tissue as a result of the diminished oxygen and nutrition supply would have been irreversible. This thesis was well borne out in the reports of De Bakey and Simeone⁴ and others.^{1 17} The incidence of amputation following arterial wounds treated within 10 hours was 36.7 per cent. In those treated from within 10 to 20 hours, it was 48.8 percent. After 20 hours, the incidence of amputation almost doubled, reaching 63 per cent.

Previous reports have indicated that in World War II the time-lag averaged over 12 hours.⁴ Half of that was the time consumed between wounding and initial tagging. This portion of time was usually influenced by the tactical situation and was thus beyond the control of the Medical Department. In an attempt to decrease the time-lag, surgical care was brought as close to the front lines as possible. In the Korean conflict, improvement was brought about by the use of helicopter evacuation. Even this was limited, since the helicopter can operate only during daylight hours and under favorable weather conditions.

During the period covered by this report, a record was kept of the time-lag in 33 repaired major vascular injuries. A comparison was also made of the time-lag in relation to the incidence of amputation. The time from injury to operation averaged 9.8 hours, a little over 2 hours less than the average in World War II. This varied greatly with the tactical situation, the terrain, and the type of fighting.

Figure 4 shows that the majority of patients were treated between 3 and 12 hours, with the largest single group being treated in from 6 to 9 hours. None of the patients treated within 9 hours required

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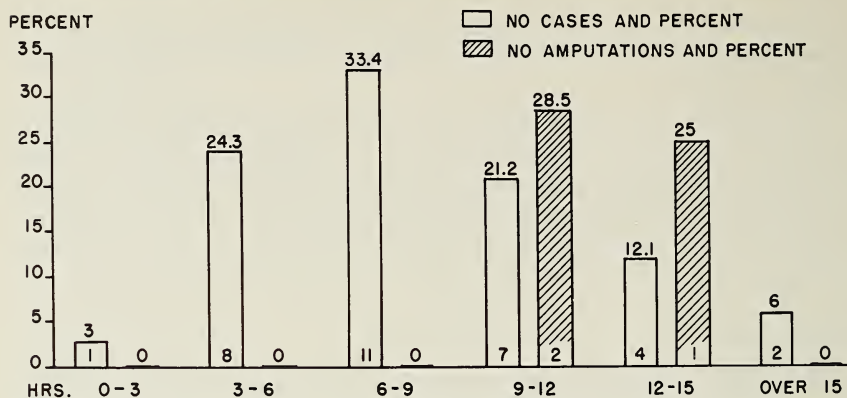


FIGURE 4. Time-lag (injury to repair) and incidence of amputation in 33 major vascular repairs.

amputation. In the 9- to 12-hour group, 28.5 per cent required amputation, and in the group treated in from 12 to 15 hours, the incidence was 25 per cent. Thus it might be concluded that the best results will be obtained in those patients treated within 9 hours. However, even in the group treated within 9 to 15 hours, at least 75 per cent could be successfully repaired. Two patients operated upon after 15 hours had severed arteries; one received treatment 18 hours after injury, and the other 48 hours after injury. Without doubt these two patients had sufficient collateral circulation to maintain the viability of the limb. By repairing the artery, however, the patients had excellent functional as well as viable limbs.

The inference to be gained from these figures is that time-lag alone is not the basis for the success or failure of vascular repair. Many of the previously mentioned factors must also be considered in the end result.

Use of the Tourniquet

The tourniquet is probably one of the most valuable lifesaving implements available to the soldier immediately following an injury of a vascular nature. It has been improvised from everything from a shoestring to a strip of the soldier's jacket or pants. In many cases, though poorly applied by inept persons, it has stemmed the flow of blood until more adequate first aid could be given by a trained medical corpsman. Despite its value, however, the use of the tourniquet is fraught with many hidden dangers. There are no statistics available to indicate the influence of the tourniquet on the incidence of amputation following a vascular wound, especially if it is left in place for long periods of time.

Many reports on the use of the tourniquet emphasize its hazards. The syndrome of profound shock following its release is well known, thus it should never be released unless adequate whole blood is avail-

able to combat vascular collapse. Intermittent release is also dangerous in that even the small amounts of blood lost at such times are not well tolerated by an already depleted vascular tree. Certainly all would agree that the thesis of "better to lose a limb than a life" is basically sound. Experimental work has shown that a tourniquet can be applied to a limb for as long as 8 hours without its resulting in permanent damage.^{2 15} Its value in traumatic amputation is well recognized. However, observations made during the treatment of this series of cases seem to indicate that the use of the tourniquet has some bearing on the results obtained, it could be replaced in the majority of cases by traction to the extremity and the application of a pressure dressing. This was possible even when the blood volume had been restored and the severed ends of the vessel were still open.

Of those patients with major vascular injuries, only 8 (23.5 per cent) had tourniquets in place at the time of arrival at the Mobile Army Surgical Hospital. Bleeding was adequately controlled after removal of the tourniquet in the manner mentioned previously. It was believed significant that all patients who later required amputation gave histories of prolonged application of the tourniquet.

There is a tendency toward the development of thrombosis in a wounded extremity. Despite the destruction of the major vascular channel, many small collaterals still function. To place a tourniquet on an extremity under these conditions not only occludes the main artery, but also occludes all collateral vessels enroute to the distal limb. These collaterals, it is true, may not be sufficient to nourish the limb, but they at least keep some blood flowing through the vessels and may impede the tendency toward thrombosis. On this basis, the application of a pressure dressing, or, when possible, the actual clamping of the involved vessel is recommended as soon as the patient is seen by the first medical officer. This will usually be at the battalion aid station.

Methods of Treatment

A standard method was used in the treatment of all vascular injuries. Lesions of minor arteries were ligated unless it was obvious that this procedure would result in arterial insufficiency and gangrene. There were 43 cases with minor vessel injury of which 41 were ligated. Repairs were performed on the two lesions of the posterior tibial artery, one by transverse suture and the other by direct end-to-end suture anastomosis. In both of these cases it was believed that ligation would have been followed by amputation.

All major vascular injuries were repaired without regard to the size of the defect, the length of time between wounding and operation, or to any other factor which could adversely affect the result.

Three types of repair were utilized. Transverse suture anastomosis was carried out in three cases, or 8.6 per cent. This was employed only if the defect in the vessel wall was quite small, involving less than 10 per cent of its circumference. The majority of cases, 28 (80 per cent), were repaired by the direct end-to-end anastomosis. In the remaining four cases (11.4 per cent), arterial continuity was re-established by autogenous vein grafts (Table 2). Arterial homo-grafts were not used, since previous personal experience had indicated that vein grafts were more satisfactory.⁵ In addition, no case was seen in which adequate sized veins were not readily available for use.

Table 2. Type of Repair in 35 Vascular Injuries

Repair	Number of Cases	Per Cent
Transverse suture	3	8.6
Anastomosis	28	80.0
Vein graft	4	11.4

Technic of Repair

As early as 1907, many of the basic principles of successful vascular repair were thoroughly described by Alexis Carrel.³ He emphasized the importance of not having any foreign material within the lumen of the vessel at the site of anastomosis, especially strands of adventitia which are most likely to produce thrombosis. He also advised against the use of smooth crushing clamps on the arterial wall because of their tendency to cause intimal damage and again predispose to thrombosis. These facts, recognized many years ago, were firmly adhered to in all the vascular repairs performed.

The authors wish to emphasize the approach used in the vascular injuries. In all cases the location of the soft-tissue wound of the extremity was ignored. Accurate appraisal was made as to the probable location of the vascular injury. The classical approaches described by Henry¹² and by Elkin⁸⁻¹¹ were employed to expose the involved segment of artery. The initial wound was extended if it lay in this course. If not, a separate incision was used for the vascular repair. This served a threefold purpose. (1) It enabled rapid exposure and accurate dissection of the involved artery on both sides of the damaged segment. When such lesions were approached through the initial wound, the extensive damage, loss of normal relationships, extravasation into the tissues, and retraction of the ends of the artery made the operation difficult and time-consuming. (2) It permitted initial control of the damaged vessel and prevented extensive hemorrhage during the remainder of the operation. In lesions approached

without this preliminary vascular control, hemorrhage was always excessive; impending shock was a continuous threat; and lower nephron nephrosis was a dangerous possibility. (3) The classical approach to the involved artery gave better exposure and made the suture repair technically easier and more rapid.

One facet of the problem of vascular repair caused much concern. As previously mentioned, the cavitational effect of the initial injury was known to cause damage far beyond the actual tract of the wounding missile. The question arose as to just how extensive this damage was in the involved artery. Certainly it could be assumed to have extended further than those areas grossly visible. To have repaired a major artery after removing only the grossly damaged segment would have been courting disaster. This may, in part, account for the very poor results of vascular repair reported from World War II as well as from the early days of the Korean conflict. To study this problem, at least 1 cc. of apparently normal vessel was excised from each end in addition to removing all grossly injured areas. This technic converted small defects into large ones and made large defects of more frightening size.

Histologic sections were made of the apparently normal vessel removed. These were stained by hematoxylin and eosin and by elastic tissue technics. It was surprising to find that in all specimens, microscopic evidence of damage of various types was found even in the most proximal areas of an apparently normal artery. Almost every case showed irregular thickening and fragmentation of the internal elastic membrane with areas of intimal loss and incipient thrombus formation. Some areas showed actual herniation of the media through the fragmented areas. Occasionally there were complete tears into the media with thrombosis in the wall of the vessel. Closer examination often revealed bleb formations on the external surface of the internal elastic membrane, an even more minute indication of injury. Though microscopic in nature, this was indisputable evidence of damage far beyond the actual site of injury.

Just how much of this damage could have resulted in thrombosis is difficult to estimate. It can be assumed that damage was present in the vessel at the site of repair and probably for an unknown distance proximally and distally. From the results obtained, this apparently did not cause thrombosis. Many of the areas examined under the microscope showed definite evidence of incipient thrombosis which probably would have caused the vascular repair to fail had they not been excised. This was obviously an important part of the technic of surgery, namely, the excision of a segment of apparently normal vessel from each end of the injured artery. The length was set arbitrarily at 1 cc. Future work may require revision of this figure. It is, of necessity, a phase of the over-all problem which deserves much basic study.

After débridement of the artery had been performed, it was apparent that simple anastomosis *per se* was impossible. Yet anastomosis was preferred over the vein graft, since it was felt that there was less chance of thrombosis if only one suture line was present. Therefore, the decision was made to sacrifice obvious important collateral branches and to strip the artery of adventitia for long distances, both proximally and distally, if this would permit end-to-end apposition. Some vessels were stripped for distances up to 10 inches. Care was taken to ligate all branches flush with the main channel to avoid possible thrombosis in those areas. Partial flexion of the extremity was also utilized for relaxation of the vessel. Elbow joints were flexed to a 90-degree angle and knee joints to a 135-degree angle. In this manner, only four cases were encountered where the final defect had to be bridged by a vein graft.

Potts coarctation clamps were placed on the cut ends of the artery to control the blood flow. The interdigitating-toothed blades of the clamps caused no intimal damage, and thus the possibility of later thrombosis was decreased. The clamps also allowed moderate traction to be exerted on the vessel which permitted easier apposition.

The anastomosis was performed by first bisecting the vessel with two interrupted everting mattress sutures. An intima-to-intima repair was then completed, using a continuous everting mattress suture of 00000 arterial silk. Leaks at the suture line were controlled by gauze pressure for several minutes or by a simple through-and-through suture.

Care was always taken to completely cover the line of anastomosis with muscle or fascia. This covering tissue was never tightly closed. In most areas, the natural elasticity of the tissue and the pressure of the sterile dressing were sufficient to attain satisfactory coverage.

The technic employed when inserting a vein graft was essentially the same except that two suture lines were necessary. In addition, the direction of the graft was reversed so that any valves present would not interfere with the arterial blood flow. All grafts were taken from the cephalic or saphenous veins, since the walls of these vessels were much thicker than those of the concomitant veins. Of necessity, lesions requiring grafts were more extensive than those in which anastomoses were done. In spite of this, it was always possible to completely cover the graft with viable muscle tissue.

The greatest problem arose in the instance of a 5-inch defect in a brachial artery. A 3-inch graft from the cephalic vein of the same arm was inserted. Because of extensive tissue destruction, the entire graft was exposed. Coverage and support were obtained by tunneling the upper part of the graft through the remnants of the biceps muscle. The flexor group of muscles of the forearm were then freed and rotated to cover the lower part of the graft, with the elbow flexed to approximately 90 degrees.

Not a single case was seen in which it was felt impractical or impossible to carry out vascular repair or to cover that repair when completed.

Results of Treatment

Of the 43 minor vascular injuries treated, gangrene occurred in five patients (11.6 per cent) requiring amputation. At the time of amputation there was evidence of thrombosis in the remaining vessels of the limbs. This was particularly interesting in a patient in whom the *profunda femoral* artery was ligated. The main femoral channel was observed at the time of débridement and was found to be grossly normal. Within 24 hours it was obvious that the limb would not survive. At the time of amputation, the femoral artery was found to be thrombosed. This was not an unusual situation. A report from the 406th Medical General Laboratory showed that in 30 per cent of a group of limbs amputated following vascular insufficiency, thrombosis was found in the apparently uninjured vessel. These cases might have represented microscopic injury as previously indicated. Gangrene might also have been the result of persistent vascular spasm or a combination of spasm and microscopic arterial injury. No investigations have been carried out to evaluate sympathectomy or anticoagulant therapy in such cases. It would be most difficult to decide which cases would be candidates for this type of therapy until thrombosis actually occurred. However, one might expect a higher incidence of thrombosis in patients with multiple wounds of the extremity.

The two cases in which the posterior tibial artery was repaired resulted in amputation. In both, the anastomoses were functioning when the limbs were removed. In one case the absorption of toxic products from the limb was the basis of amputation. In the other patient the limb was viable, but the extensive bone, nerve and soft-tissue loss made it useless and a midcalf amputation was made approximately 3 weeks after the initial injury.

Reports from World War II, and the results of this series, would seem to indicate that little improvement can be expected in the treatment of minor vascular injuries. Ligation continues to remain the treatment of choice. In very selected cases, repair of the posterior tibial artery may be indicated, since the anterior tibial and the peroneal arteries are relatively small and much may depend upon continuity of the posterior tibial.

The results obtained in the 34 major vascular injuries treated were far more encouraging, with an amputation rate of but 8.8 per cent (three cases, Table 3). Thirty-three arteries were repaired by the methods previously described. One brachial artery, severed just above the elbow, was ligated. The retrograde flow was equal to that of the proximal segment and there was no advantage in repair.

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Table 3. Comparison of the Incidence of Gangrene in Major and Minor Vascular Injuries

Vessels	Number of Cases	Per Cent	Gangrene	
			No. Cases	Per Cent
Major	34	44.2	3	8.8
Minor	43	55.8	5	11.6

The authors were impressed that, of the 77 cases treated, the incidence of amputation was actually higher in those cases in which minor vessels were ligated than in those where major vessels were repaired. No definite explanation could be made for this most unusual result. If the basis of amputation in the minor vascular lesions was thrombosis, possibly this factor may have been of less significance in the major vascular lesions. It has been shown experimentally that thrombosis is more prone to occur in injury to the smaller arteries. This factor, together with the unusually high incidence of successful repairs, may in part account for this reversal of the expected ratio.

Two of the seven patients with popliteal artery repairs (28.5 per cent) later required amputation of the involved limb. Among a total of nine patients having repair with a vein graft in the femoral artery, one patient required amputation. This was an incidence of 11.1 per cent. In the remaining 30 patients with major vascular repairs, no limbs were lost (Table 4).

Table 4. Results in 33 Major Arteries Repaired

Vessel	Number of Cases	Per Cent	Treatment			Loss of Limb	
			Anastomosis	Vein Graft	Transverse Repair	Number Cases	Per Cent
Carotid	1	3.0	1	-----	-----	-----	-----
Axillary	5	15.2	4	1	-----	-----	-----
Brachial	10	30.4	9	(1)	-----	-----	-----
above profunda	4	12.2	4	-----	-----	-----	-----
below profunda	6	18.2	5	1	-----	-----	-----
External iliac	1	3.0	1	-----	-----	-----	-----
Femoral	9	27.2	6	(2)	(1)	(1)	11.1
above profunda	1	3.0	1	-----	-----	-----	-----
below profunda	8	24.2	5	2	1	1	12.5
Popliteal	7	21.2	6	-----	1	2	28.5
Total							
Average	33	100	27	4	2	3	9.1

There was only one known thrombosis at the suture line. This occurred in a patient who had both the femoral and popliteal arteries severed in the same limb. On admission, he was in profound shock having had a tourniquet on the involved limb for over 6 hours. Only the femoral artery injury was recognized initially. At the time of operation, which was 10 hours after injury, an end-to-end anastomosis was performed in the femoral area. A total of 2 inches of femoral artery was excised. During débridement of the other wounds of the leg, the severed popliteal artery was recognized. Because of the large segment of artery excised in the femoral area, only grossly damaged vessel was removed in the popliteal area so that an anastomosis could be performed. Postoperatively, the patient had a good posterior tibial pulse at the ankle on the involved side. He developed a lower nephron syndrome which was successfully treated by two "runs" on the artificial kidney. On the fourth postoperative day, however, it was obvious that the limb was necrotic. At the time of amputation, the popliteal anastomosis was occluded. The femoral anastomosis was found to be patent.

Many factors were present which could have accounted for this thrombosis. The fact that there were two lesions in the same limb, of which only one failed, permitted us to use the successful femoral repair as a control. On this basis, the one outstanding difference in treatment was the lack of excision of a cuff of relatively normal artery in the popliteal region. In retrospect, we would have excised a cuff and inserted a vein graft. This case helped to confirm our impression that a generous segment of vessel must be removed prior to repair. The profound shock, the time-lag of 10 hours, and the lower nephron syndrome were also complicating factors which could have resulted in failure. We felt they could be discounted, since they had no apparent effect on the femoral repair.

A most unusual situation was encountered in the two remaining cases of a vein graft of the femoral artery and a popliteal anastomosis which terminated in amputation. Both had had tourniquets applied to the limb for long periods of time. Both had some degree of vascular collapse which was rapidly overcome with whole blood transfusions. The time-lag in the former was 10 hours, and in the latter, 14 hours. Following operation, the limbs which had been cold, firm and pulseless, became warm; and good arterial pulsations were felt at the ankle. It was expected at the time that both limbs would survive.

The patient with the popliteal anastomosis developed dry gangrene of the tips of the second, third, and fourth toes. This did not progress. On the 14th postoperative day, because of some initial signs of toxicity, it was decided to make an exploration of the wound; and the toes had to be amputated. At that time the patient still had excellent arterial pulsations at the ankle. On exploration, it was obvious that the muscles of the leg were nonviable. However, on cutting across the calf muscles, normally pulsating arterial blood flowed from all the major arteries and the large arterioles. The anastomosis was still

functioning and was, therefore, left in the limb. Amputation was performed below the knee.

The patient with the vein graft in the femoral artery showed an almost identical picture. He was re-explored on the eighth postoperative day for early signs of toxicity. Dry gangrene was observed in the tips of the great and second toes. Again good arterial pulsations were present at the ankle, and during operation arterial blood pulsated from all the major arteries of the leg and from the large arterioles. A supracondylar amputation was performed and the vein graft was removed for examination. There was no evidence of thrombosis at the suture lines. Microscopic sections of the graft revealed the usual extensive intimal proliferation and active fibroblastic growth. There was also active connective tissue growth in the media, and the internal elastic membrane was intact.

It is believed that these two failures were due to probable thrombosis of the small arterioles and capillaries in the muscle which prevented the oxygenated blood from reaching the tissue cells where it was needed. Possibly the long period of initial anoxemia and ischemia prior to restoration of the blood flow, caused the irreversible changes in the tissues and smaller vessels of the limb.

It is important to emphasize, however, that in both cases the vascular repairs were successful and functioning. Had these patients been treated earlier, they probably would have maintained a viable limb. These cases are also important in that they demonstrate the interplay of the many factors referred to previously in this report which help determine the ultimate results of treatment.

It was obvious that some of the major vascular lesions which were repaired could have been ligated without fear of subsequent gangrene and amputation. Based on past reports, it was felt that utilization of reparative surgery in all major vascular lesions would not only decrease the incidence of amputation, but, of more importance, would result in a high degree of functional, as well as viable, extremities.

Late Follow-up

It has been possible to obtain late follow-up studies on 18 of the 33 major vascular injuries repaired. Patients who had no associated bone or nerve damage were returned to military duty, not having left the Theater of Operation. This in itself was evidence of the excellent functional results obtained by arterial repair. Fourteen, or 42.4 per cent, of the patients with major vascular repairs were returned to the Zone of Interior and Walter Reed Army Medical Center because of associated injuries. Complete vascular surveys were made on this group.

Excellent peripheral arterial pulsations were present in all but two patients. Both of these had brachial artery anastomoses. Even in these patients the pulses were present, but weak. One was explored because of median nerve damage and the anastomosis was seen and known to be functioning though moderately constricted. The other

patient was being treated for hepatitis, thus delaying further study.

Skin temperature studies and oscillometric studies have been done and have consistently shown readings on the repaired limb equal to those on the normal side. The only exceptions were in the cases of the two brachial repairs. Here the readings were depressed. Functionally, there has been no evidence of arterial insufficiency.

Probably the most important and convincing evidence of patency of the repair is an arteriogram. Whenever possible, this procedure has been carried out. Three patients, on whom neurolyses were performed and the anastomoses were found to be functioning, were not subjected to the further discomfort of an arteriogram. In all patients in whom this procedure was carried out, the repairs were observed to be patent. Slight constriction was noted at the suture line in some cases. This was observed in all our experience with arterial repairs. In other cases it was impossible to see the actual suture. A conical type of constriction was noted in a femoral artery just distal to a vein graft, but this apparently did not interfere with its function. It may have resulted from scar-tissue formation, but it is more likely due to tension or constriction at the distal suture line, a technical fault which could have been avoided.

Of the group of foreign patients with major vascular repairs, which constituted only a small percentage of the entire group, follow-up has been by correspondence. There have been no losses of limbs or complaints of symptoms which could be considered due to arterial insufficiency. Excellent palpable distal pulses have been described in all limbs.

It is possible that these vascular repairs may become occluded at some later date. Based on previous extensive experience with anastomoses and grafts, we do not feel that this will occur. But should it happen, the patient will certainly not lose a limb. The follow-up in this series covered a period of only 4 to 7 months. It is firmly believed that the repair of the acute vascular injury is of unquestionable value and should be carried out in all cases of major arterial damage.

Summary

This report included the treatment and results obtained in 77 consecutive acute vascular lesions occurring in war wounds in Korea. Many of the factors influencing the results obtained are discussed in relation to this group of cases. Forty-three minor vascular injuries were treated by simple ligation of the artery. Amputation of the involved limb was later necessary in five patients, or 11.6 per cent. Thirty-four major vascular lesions were repaired by direct end-to-end suture anastomosis or autogenous vein graft. Amputation was later necessary in only three (8.8 per cent) of the patients in this group. Repair is recommended in all cases of acute major arterial injury if the incidence of amputation is to be decreased, and if a functional as well as viable limb is to be obtained.

References

1. Bradford, B., Jr.: Vascular Injuries in War. *Surg., Gynec. & Obst.* 83: 667, 1946.
2. Campbell, D.: Walter Reed Army Medical Center, Washington 12, D. C. Unpublished data.
3. Carrel, A.: The Surgery of Blood Vessels. *Johns Hopkins Hosp. Bull.* 18: 18, 1907.
4. De Bakey, M. E., and Simeone, F. A.: Battle Injuries of the Arteries in World War II. *Ann. Surg.* 123: 534, 1946.
5. Cooke, F. N.; Hughes, C. W.; Jahnke, E. J., Jr.; and Seeley, S. F.: Homologous Arterial Grafts and Autogenous Vein Grafts Used to Bridge Large Arterial Defects in Man. Presented at the Society for Vascular Surgery, Chicago, June 8, 1952.
6. Elkin, D. C.: Vascular Injuries of Warfare. *Ann. Surg.* 120: 284, 1944.
7. ———: The Treatment of Aneurysms and Arteriovenous Fistulas. *Bull. New York Acad. Med.* 22: 81, 1946.
8. ———: Arteriovenous Aneurysms. *J. A. M. A.* 129: 26, 1945.
9. Elkin, D. C., and Cooper, F. W., Jr.: Resection of the Clavicle in Vascular Surgery. *J. Bone & Joint Surg.* 28: 117, 1946.
10. Elkin, D. C., and Harris, M. H.: Arteriovenous Aneurysm of the Vertebral Vessels. *Ann. Surg.* 124: 934, 1946.
11. Elkin, D. C., and Kelly, R. P.: Arteriovenous Aneurysm. *Ann. Surg.* 122: 529, 1945.
12. Henry, A. K.: Extensile Exposure Applied to Limb Surgery. Williams and Wilkins Co., Baltimore, 1945.
13. Howell, W. H.: Mobile Army Surgical Hospital 8055 Army Unit. Personal communication.
14. Makins, G. W.: Gunshot Injuries to the Blood Vessels. John Wright and Sons, Ltd., Bristol, 1919.
15. Miller, H. H., and Welch, C. S.: Quantitative Studies of the Time Factor in Arterial Injuries. *Ann. Surg.* 130: 428, 1949.
16. Odom, C. B.: Causes of Amputation in Battle Casualties with Emphasis on Vascular Injuries. *Surgery*, 12: 488, 1942.
17. Rose, C. A.; Hess, O. W., and Welch, C. S.: Injuries of the Extremities in Battle Casualties. *Ann. Surg.* 123: 161, 1946.
18. Russell, J. P.: Secondary Hemorrhage in War Wounds. Symposium on Military Medicine in the Far East Command. September, 1951.
19. Seeley, S. F.; Hughes, C. W., and Jahnke, E. J., Jr.: Direct Anastomosis Versus Ligation in Traumatic Arteriovenous Fistulas and Aneurysms. *Surgical Forum*, American College of Surgeons, New York City, September 23, 1952.
20. Seeley, S. F.; Hughes, C. W.; Cooke, F. N., and Elkin, D. C.: Traumatic Arteriovenous Fistulas and Aneurysms in War Wounded. *Am. J. Surg.* 83: 471, 1952.
21. Seeley, S. F.; Hughes, C. W., and Jahnke, E. J., Jr.: Unpublished data.
22. Shumacher, H. B., Jr., and Carter, K. L.: Arteriovenous Fistulas and Arterial Aneurysms in Military Personnel. *Surgery*, 20: 9, 1945.
23. Simeone, F. A.: Notes on Treatment of Wounds of the Arteries. Symposium on Military Medicine in the Far East Command. September 1951.
24. Smith, M. K.: Arterial Injuries. *Ann. Surg.* 126: 866, 1947.
25. Steward, C. F.: War Experiences with the Non-Suture Technic of Anastomosis in Primary Arterial Injuries. *Ann. Surg.* 125: 157, 1947.
26. Warren, R.: Report to The Surgeon General, Department of the Army. Washington 25, D. C., April 1952.

Chapter 9

Arterial Injuries in the Korean Conflict:

Experiences with 111 Consecutive Injuries*

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Among the advances made by medical officers in the Korean conflict, none were more dramatic than the successful repair of acute arterial injuries. This report is a summary of experiences at a forward surgical hospital during the last 18 months of the conflict. It was during this period that the technic of arterial repair of the battle wound was developed.¹⁻³ As a result, this report spanned and contrasted two periods, the period of arterial ligation and the period of arterial repair (Table 1).

Table 1. Summary of Method of Management of Acute Arterial Injuries

Method of Management	Number
Ligation: January to April 1952	26
Restoration of continuity: April 1952 to June 1953	75
Observation (common carotid)	1
Died of uncontrolled hemorrhage (iliac)	9
Total	111

Prior to April 1952, ligation of transected arteries was almost routine. The disastrous results are well known.^{4,5} Twenty-six major peripheral arteries were ligated, 24 of which were in consecutive casualties, with a resultant amputation rate of 62 per cent (Table 2). In two of these patients, the common carotid artery was also ligated and both developed a hemiparesis. Following ligation, two or more additional patients developed distal ischemia to such an extent that the function of the limb was seriously impaired.

* Previously published in *Surgery* 37: 850, 1955.

Table 2. Results of Ligation

Artery	Total Number	Type of Repair					
		Satisfactory		Ischemia with Marked Functional Impairment		Amputation	
		Number	Per Cent	Number	Per Cent	Number	Per Cent
Subclavian	1	-----	-----	-----	-----	1	100
Axillary	4	1	25	2	50	1	25
Brachial	6	3	50	-----	-----	3	50
Common femoral	3	1	33	-----	-----	2	67
Superficial femoral	4	1	25	-----	-----	3*	75
Popliteal	6	-----	-----	-----	-----	6	100
Common carotid	2	-----	-----	2**	100	0	0
Total	26	6		4		16	
Over-all per cent		23 %		15 %		62 %	

*Hunter's canal.

**Hemiparesis.

The high rate of amputation following ligation has therefore not diminished since De Bakey and Simeone reported the results of ligation in World War II (Table 3).⁴

Because of this high rate of major amputations resulting from arterial ligation—based on the progress made in vascular surgery since World War II—primary repair of arterial injuries was undertaken as a formal study by the Surgical Research Team in Korea during 1952.^{1,3,6} Thereafter, at the 46th Surgical Hospital 75 acute arterial injuries were repaired primarily. These casualties represented consecutive arterial injuries, with the exception of two soldiers who could not be resuscitated sufficiently to permit adequate operation and a third soldier who was semicomatose following an injury to the common carotid. The latter casualty was observed and evacuated without operation. Therefore, patients were unselected, except for the crude selection based on the ability of the patient to survive operation. With this exception, all major arteries were repaired regardless of the time lag or ischemic changes in the distal extremity.

Improvement in the results was unequivocal. The amputation rate fell from 62 per cent following ligation to 7 per cent following repair (Fig. 1; see Table 3). A fall in the amputation rate was noted following injuries to the upper and lower extremities (Fig. 2) as well as following injury to each of the major vessels (Fig. 3; Table 4).

Arterial Injuries in Korea

Table 3. Percentage of Amputation After Arterial Injuries

Artery	World War II		Korea			
	Num- ber	Percent- age Ampu- tated	Ligation		Repair	
			Num- ber	Percent- age Ampu- tated	Num- ber	Percent- age Ampu- tated
Subclavian	21	29	1	100	1	0
Axillary	74	43	4	25	4	0
Brachial			6	50	25	0
above profunda	97	56				
below profunda	209	26				
Internal iliac	1	0			1	0
External iliac	30	47			3	33
Common femoral	106	81	3	67	9	0
Superficial femoral	177	55	4	75	12	8
Popliteal	502	73	6	100	17	18
Common carotid			2	100*	3	0
Total			26		3	
Average				62		0

*Hemiparesis not included in over-all amputation rate of 62 per cent.

Table 4. Results Following Restoration of Arterial Continuity

Artery	Total Number	Type of Result			
		Satisfactory		Amputation	
		Num- ber	Per Cent	Num- ber	Per Cent
Subclavian	1	1	100	0	0
Axillary	4	4	100	0	0
Brachial	25	25	100	0	0
Internal iliac	1	1	100	0	0
External iliac	3	2	67	1	33
Common femoral	9	9	100	0	0
Superficial femoral	12	11	92	1	8
Popliteal	17	14	82	3	18
Common carotid	3	3	100	0	0
Total	75	70		5	
Average			93%		7%

The Battle Wound: Clinical Experiences

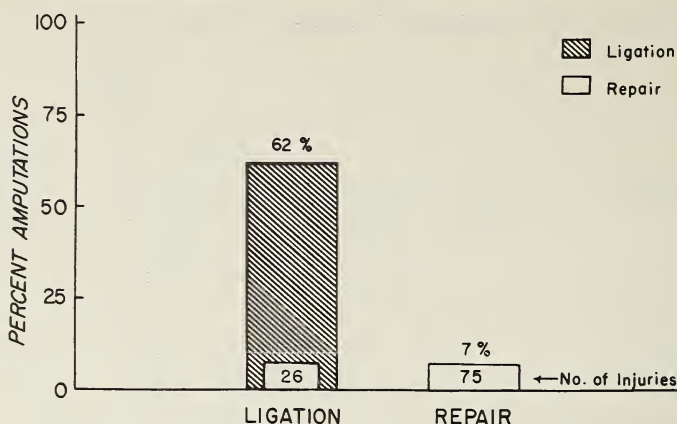


FIGURE 1. Demonstrating the decrease in the amputation rate following the institution of arterial repair.

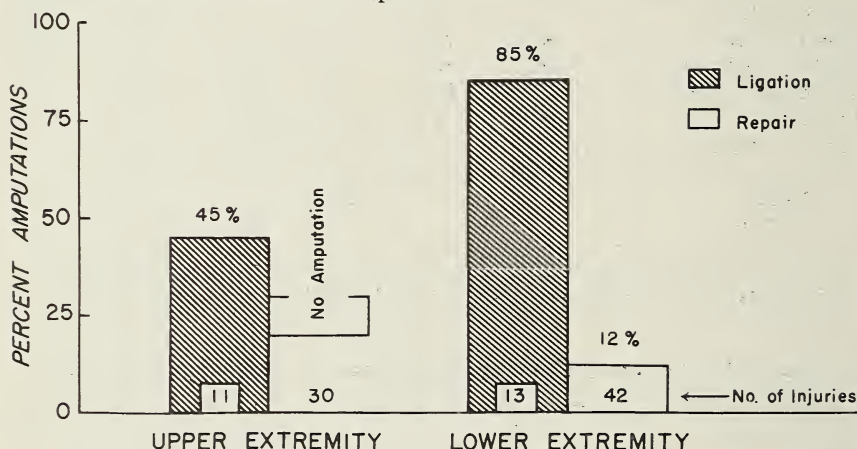


FIGURE 2. Following arterial repair, amputation of the upper extremity was unnecessary. All five amputations following arterial repair occurred with wound of the lower extremities.

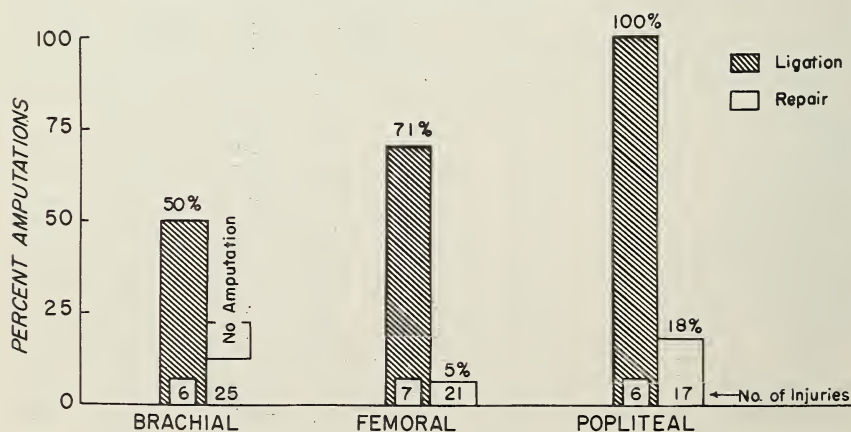


FIGURE 3. A striking reduction in the amputation rate was noted following the repair of each of the major arteries.

Surgical Management

Diagnosis

The diagnosis of an arterial injury may be completely clear-cut or tantalizingly equivocal. It became obvious in our experience that a high index of suspicion was the greatest aid in the preoperative diagnosis of arterial injury. In spite of this, the diagnosis frequently was made only at operative exploration of the vessel in question.

As a rule, inspection will reveal two or three points aiding in the diagnosis. The location of the wounds of entrance and exit should lead one to suspect the possibilities of vascular injury. Following vascular injury, the relative size of the involved extremity is sometimes greater than normal and the swelling is sometimes tense. The extremity may be pale or it may have a ruddy cyanotic appearance; the former appearance indicates arterial injury and the latter perhaps both arterial and venous involvement.

Auscultation may occasionally reveal a bruit over the area of injury. Blood pressure differentials and oscillometric readings were not determined in this study.

The temperature differential is generally of diagnostic aid, but it becomes of no value in a soldier who has been lying out in subzero weather for several hours before being picked up. Absence or diminution of peripheral pulsation falls in this same category. Both lowered temperature and decreased pulsation may be present in an extremity whose major vessel has no organic injury but whose circulation is impaired by a displaced bone fragment, segmental spasm, or generalized peripheral vascular collapse. However, such an extremity must be explored if the wound lies in the proper position to have injured an artery. Of equal importance in the diagnosis is the unequivocal fact that a pulse often may be palpated distant to the site of an arterial injury.

Examination of the roentgenograms and study of the probable missile path will frequently give a clue as to the presence or absence of arterial injury. As stated previously, it is necessary to explore a vessel simply on the basis of probability determined by the position of the retained missile or path of the wound. This statement is true, even in the absence of any or all of the diagnostic signs of arterial injury; for all too frequently an arterial injury becomes evident only when the wounds are more adequately explored and débrided.

Principles of Treatment

The first principle, that restoration of continuity must be achieved in order to save extremities, is dependent upon several time-worn but oft-ignored secondary principles. Exposure must be adequate; and it should be achieved before any clot in the vessel is dislodged.

Once exposure and hemostasis are obtained, the success of the anastomosis depends on several factors. (1) Débridement of the soft tissues must be adequate, as infections in inadequately débrided wounds may lead to secondary thrombosis or secondary hemorrhage. (2) The defective artery itself must be adequately débrided, as intimal damage may lead to early thrombosis. Resection of the artery should include all grossly damaged areas. (3) As previously described by others,^{3 5 7 8} anastomosis should result in a smooth intima-to-intima approximation. (4) Anastomosis must be achieved without tension on the suture line. The success of the operation is primarily dependent upon the application of this technical discipline. Short cuts will lead to thrombosis. Grafts from the cephalic or saphenous vein were used in 16 casualties with results which, within the limits of this study, appeared completely satisfactory (Table 5). The insertion of a vein graft is far superior to anastomosis with tension.

**Table 5. Results of Repair of Arterial Injuries
(72 Patients and 75 Injuries)**

Artery	Number of Arteries	Type of Repair			Amputations
		Anasto- mosis	Trans- verse Repair	Vein Graft	
Subclavian	1	0	0	1	
Axillary	4	3	0	1	
Brachial	25	19	4	2	
Iliac	4	3	0	1	1 following anastomosis
Common femoral	9	3	2	4	1 following anastomosis 2 following anastomosis 1 following vein graft
Superficial femoral	12	8	2	2	
Popliteal	17	10	3	4	
Common carotid	3	0	2	1	
Total	75	46	13	16	

Under the conditions in Korea, a prolonged time lag before operation was not a contraindication to repair. Successful re-anastomosis was achieved as late as 20 hours after injury (Table 6). To date, observations suggest that surgical failure is dependent upon technical errors at the time of repair or ischemic changes in the distal muscle prior to operation. As previously described by others,^{3 6 7} an anastomosis may be technically perfect and the distal arterial pulsation full; yet gangrene of the muscles may require amputation. This clinical picture was noted twice. The skin was viable and the distal

pulse was excellent; yet the muscles were dead. Although specific data is not yet available, this is believed to be the result of preoperative ischemia due to arterial interruption rather than to small vessel thrombosis and continued postoperative ischemia. As in the case of incarcerated hernias, time lag prior to surgery has a relative significance rather than an absolute significance. If collateral circulation is good, a successful anastomosis may be achieved after many hours for the success, in this respect, seemingly does not depend upon changes in the artery itself but in the muscle. Conversely, following the complete interruption of the circulation for only a few hours, restoration of the arterial flow may occasionally prove futile.

Table 6. Time Lag Before Restoration of Arterial Continuity

Time (hours)	Number of Patients*	Number of Amputations	Percentage Amputated
0-3	2	0	0
3-6	24	2	8.0
6-9	25	0	0
9-12	16	2	12.5
12-15	4	1	25.0
over 15	4	0	0
Total	75	5	
Average time-lag-----	-----	-----	7.9 hours

*Three patients with two injuries are included twice.

Postoperative Complications

There were two types of infections encountered in this group of patients; the first was gas gangrene involving the peripheral portions of the extremities. These patients were those in whom the surgeons took a chance on an extremity that should have been amputated originally. The second type of infection was that developing at the site or around the site of the anastomosis (Table 7). In all probability, this was primarily due to inadequate débridement of the wound. The presence of these wound infections did not necessarily result in a breakdown of the repair in every instance. However, this was the most common outcome. Secondary repair was attempted twice; but success was doubtful as far as the anastomosis was concerned. Amputation was unnecessary in these latter two patients; apparently the delay in occlusion was sufficient to permit development of adequate collateral circulation to maintain a viable and functionally useful limb.

Table 7. Major Complications of Arterial Repair

Leading to amputation:	
Primary thrombosis	2
Wound infection with secondary thrombosis	1
Preoperative ischemia (anastomosis patent)	2
No amputation necessary:	
Secondary hemorrhage	2
Pulmonary embolus	1
Thrombosis (including follow-up)	5
Wound infection	3

Breakdown of an arterial repair occurred only in the presence of a wound infection. The development of thrombosis at the site of repair or distal to the anastomosis was infrequent. It occurred four times in the immediate postoperative period. It occurred again in a vein graft whose distal circulation was questionable at the time operation was completed. Late thrombosis is known to have occurred in four others; but the follow-up on these patients was incomplete. The latter four retained useful limbs. Sympathectomy and anticoagulants were not used following arterial repair.

Three patients in the series died, two from associated injuries and one from a pulmonary embolus which occurred 30 days following reparative surgery of other injuries.

Problems

Iliac Arteries. One of the unsolved problems is the management of injuries of intra-abdominal arteries. Patients with aortic injuries did not reach the hospital alive. Not infrequently, however, casualties with injuries of the iliac arteries reached the hospital alive only to die of continued hemorrhage prior to operation or shortly after the induction of anesthesia. The problem of arterial injuries is not solved until the problem of intra-abdominal hemorrhage is solved.

Carotid Arteries. The authors treated six patients with injuries to the common carotid artery. One patient was unconscious and considered inoperable. He was evacuated after 2 days of observation. Two patients had the carotid ligated prior to the period of reparative therapy, and both developed a severe hemiparesis. Three casualties had the vessel repaired, one with a vein graft and two with transverse repairs; and these three patients had an uncomplicated convalescence. Additional study is required before a policy of primary repair of carotid arteries can be firmly established. At present, it appears to be the treatment of choice.

Arterial Spasm. As evidenced by the frequent delay before the distal pulse returns after anastomosis, arterial spasm is not an infre-

quent sequela of trauma. Clinically significant spasm is an extremely difficult problem, although an infrequent one. In one instance, the spasm responded to wrapping the spastic segment for 15 minutes with a sponge soaked with 2.5 per cent papaverine.⁹

Two other patients were called to our attention in whom this complication occurred; and it was noted in both patients at the time of primary surgery. One did not respond to papaverine or procaine soaks. The surgeons then distended the femoral segment with intraluminal saline; but the spasm returned. Finally, the anastomosis was taken down and an additional segment of artery excised and a vein graft inserted; but this treatment was inadequate, and amputation was necessary eventually. Amputation was required in a third patient having a similar wound, in spite of lumbar sympathectomy and the use of procaine locally and intravenously. Again this is infrequent as a major complication; and it occurred only once in this series.

Conclusion

Out of the experiences in Korea, one very important general conclusion is evident, *i. e.*, that primary, restorative surgery of major arterial injuries is feasible and mandatory. The feasibility of primary repair in a forward surgical hospital has been clearly demonstrated by the fact that, following restoration of continuity in 75 consecutive wounds, the amputation rate fell from the earlier experiences of 48 and 62 per cent following ligation to 7 per cent following primary repair.

References

1. Jahnke, E. J., Jr.: The Problems and Management of Acute Vascular Injuries. Preliminary Report to the Army Medical Service Graduate School, 1952.
2. Jahnke, E. J., Jr., and Howard, J. M.: Primary Repair of Major Arterial Wounds. *Med. Bull. U. S. Army, Far East* 1: 43, 1953.
3. Jahnke, E. J., Jr., and Howard, J. M.: Primary Repair of Major Arterial Injuries. *Arch. Surg.* 66: 646, 1953.
4. De Bakey, M. E., and Simeone, F. A.: Battle Injuries of the Arteries in World War II: An Analysis of 2,471 Cases. *Ann. Surg.* 123: 534, 1946.
5. Ziperman, H. H.: Acute Arterial Injuries in the Korean War. A Statistical Study. *Ann. Surg.* 139: 1, 1954.
6. Hughes, C. W.: Acute Vascular Trauma in Korean War Casualties: An Analysis of 180 Cases. *Surg., Gynec. & Obst.* 99: 91, 1954. (Chapter 12 of this volume.)
7. Jahnke, E. J., Jr., and Seeley, S. F.: Acute Vascular Injuries in the Korean War. *Ann. Surg.* 138: 158, 1953. (Chapter 8 of this volume.)
8. Jahnke, E. J., Jr.; Hughes, C. W., and Howard, J. M.: The Rationale of Arterial Repair on the Battlefield. *Am. J. Surg.* 87: 396, 1954.
9. Kinmunth, J. B.: The Physiology and Relief of Traumatic Arterial Spasm. *Brit. Med. J.* 1: 59, 1952.

Chapter 10

The Primary Repair of Wounds of Major Arteries:

An Analysis of Experience in Korea in 1953*

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Damage to blood vessels of the extremities constitutes a small but important component of war wounds. Of 2,471 such wounds collected by De Bakey and Simeone from World War II,¹ 49.6 per cent of those with involvement of a major artery had to be amputated. Ligation of damaged vessels was the accepted practice. They further reported 81 cases in which suture of the artery was performed. In these 81 cases, the amputation rate was 35.8 per cent.

Shortly after the beginning of the Korean conflict, Walter Reed Army Hospital was designated as the peripheral vascular surgery center for the Army. This center received a substantial number of Army personnel who had sustained traumatic, arteriovenous fistulas and aneurysms. The results of reparative and reconstructive surgery of many of these lesions have been reported.²⁻⁴ On the basis of this experience, primary repair of damaged major blood vessels of extremities was considered practicable.

Personnel trained in the technic of blood vessel repair were sent to Korea as members of the Surgical Research Team of the Army Medical Service Graduate School. The results of their study established the practicability of repair of major arteries of extremities as part of the definitive surgery of war wounds. A total of 130 major vessel injuries were repaired and follow-up data were reported by three members of the Surgical Research Team (Table 1).⁵⁻⁷ The average amputation rate for these 130 vascular repairs was 10.8 per cent.

Ziperman⁸ analyzed the results of 234 major and minor arterial wounds treated in Korea during the first 9 months of 1952. His report included the work of two members of the Surgical Research Team. He compared the over-all findings in his group to the results reported by De Bakey and Simeone and he reported 127 major arteries repaired by suture, with an amputation rate of 20.5 per cent. This represents a 42.7 per cent decrease in amputation rate. Both of these series, however, are based on relatively small numbers.

*Previously published in *Annals of Surgery* 141: 297, 1955.

Primary Repair of Wounds of Major Arteries

This study includes an analysis of all major vascular wounds repaired in Korea from January 1 through July 1953 (exclusive of Navy and Marine personnel), during which time 211 major arterial injuries were detected in 205 patients. Two patients had three vascular injuries each, and two other patients had two injuries each. The author treated 72 of these vascular injuries (*see* Table 1); and they are reported in detail in Chapter 11 of this volume.⁵

Table 1. Controlled Follow-up Studies on Major Arterial Repairs in Korea, 1952-1953

Investigator	Cases	Amputations	
		Number	Percentage
Jahnke (1952)	34	3	8.8
Howard (1952)	24	3	12.5
Hughes (1953)	72	8	11.1
Total	130	14	
Average			10.8

Materials and Methods

Reports on all cases in the Korean theater were centralized in the Office of the Surgeon of the Eighth United States Army. They emanated from the forward hospitals in which the surgery was performed and from the evacuation hospitals through which these patients passed.

The results are tabulated as short-term and late follow-up studies. Such a division was necessary because this series included 61 patients who were Korean nationals or prisoners of war and other United Nations' personnel, many of whom were not available for later observation. Late follow-up studies were accomplished on other Allied patients as they returned to vascular centers in Japan and on American patients who were returned to Walter Reed Army Hospital where they were subsequently evaluated by arteriography, oscillometry, skin temperature and exercise tolerance studies.

In this over-all theater total of 211 major vascular injuries, deaths occurring from causes other than arterial injury were reported in 15 patients with 18 arterial repairs. Arteries involved in these patients were the carotid 1, axillary 1, common iliac 1, femoral (unqualified) 3, common femoral 2, superficial femoral 3, and popliteal 7. Since an adequate evaluation of the arterial repairs in these patients

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was not possible, these 18 repairs have been eliminated, leaving 193 repaired major vascular injuries in 190 surviving patients.

Regardless of efforts made to follow through on every patient, 62 of the 193 repairs in surviving patients were lost to long-term follow-up studies because of evacuation after operation to Korean or United Nations hospitals. Thus, 193 arterial injuries were available for short-term studies; and 131 of these were included in the follow-up studies.

Approximately 85 per cent of the vascular wounds were caused by fragmenting missiles and 15 per cent by nonfragmenting missiles. Of all the vessels damaged, the majority were lacerated although an almost equal number were severed. Only a few vessels were thrombosed or in spasm (Table 2). The lower extremity was the most prevalent site of vascular injury (55.5 per cent), with the upper extremity a close second (38.4 per cent), while the neck and trunk received a very low percentage of the injuries. A distribution of injuries is shown in Table 3.

Table 2. Major Vascular Injuries in Korea, 1953

Type of Injury	Number
Lacerated	113
Severed	89
Contusion and thrombosis	7
Spasm	2
Total	211

Table 3. Major Vascular Repairs in Korea, 1953

Region	Artery	Number	Percentage
Neck	Carotid	7	3.3
Upper extremity	Axillary	13	38.4
	Brachial	68	
Trunk	Aorta	1	2.8
	Common iliac	3	
	External iliac	2	
Lower extremity	Common femoral	12	55.5
	Superficial femoral	68	
	Popliteal	37	
Total		211	100.0

Data available from approximately one-third of the patients in this study showed that 40 per cent of the patients arrived in shock of varying degree, and 47 per cent had a tourniquet applied for an average of 4 hours.

Vascular injuries received a high, but not first priority evacuation unless there was uncontrolled hemorrhage or severe shock which could not be treated prior to evacuation. The time lag from injury to operation for all cases of vascular injury in 1953 averaged 9.8 hours, almost identical to that reported by Ziperman in 1952. Approximately 6 hours of this time was spent reaching the hospital, and 4 hours in preparation for surgery. This is a reflection of the degree of injury and shock in these patients.

An anatomic, surgical approach was used regardless of location of the wound. An adequate incision was always made and proximal control of the damaged vessel secured first. After excision of the damaged portion of the vessel, repair was accomplished by a continuous mattress suture with 00000 braided arterial silk everting the edges of the artery, apposing intima to intima.

Table 4. Types of Repair in Major Vascular Wounds in Korea, 1953*

Repair	Number	Percentage
Anastomosis	125	64.8
Vein graft	28	14.5
Arterial graft	11	5.7
Lateral repair	19	9.8
Conservative	4	2.1
Release spasm	2	1.0
Remove thrombus	1	0.5
Ligation	3	1.6
Total	193	100.0

*Excluding 18 repairs in patients who died.

Most of the repairs (64.8 per cent) were accomplished by direct anastomosis, with autogenous vein grafts (14.5 per cent) the second most common type of repair. Lateral suture repair followed in third place, and homologous arterial grafts in fourth place (Table 4). Conservative nonoperative treatment was practiced in some instances of nonexpanding pulsating hematomas, especially of the carotid artery. Ligation was utilized only where repair was not feasible in a noncritical artery or when the patient's condition did not permit further surgical procedures.

After careful débridement, care was taken to cover the repaired vessel in order to nourish and protect it; but the wound was left open

for drainage and to minimize the risk of infection. Penicillin and streptomycin were used routinely in all patients. Sympathectomy was not practiced, and anticoagulants were not utilized. Following operation, only those extremities with complicating fractures were placed in casts. For a period of 2 weeks, only limited active motion was permitted at the vascular repair site. If the wound remained clean, delayed closure was accomplished on the fourth to sixth postoperative day.

Results

Of the 193 vascular repairs in surviving patients, 26 required amputations. These amputations were performed following repairs of the axillary artery in 2 cases, brachial artery (unqualified) 2, femoral (unqualified) 4, common femoral 5, superficial femoral 2, and popliteal 11, most of these being considered critical arteries. The percentage of amputations was higher in those cases requiring grafts for repairs (*see* Table 5).

Causes for amputation were determined where possible. Seven patients were reported as having good blood flow through necrotic muscle at the time of amputation. The muscle had undergone irreversible changes prior to reconstruction of the artery. Thrombosis was responsible for three amputations; compression of the repaired vessel by a displaced open, comminuted fracture was responsible for one amputation, and venostasis for another. The reason for amputation in the remaining 14 was not recorded. Of these, five limbs were amputated over varying periods of several days to 3 months following repair of the damaged artery. In one case reported here as an amputation, the patient lost only four toes; while another had a transmetatarsal amputation.

An attempt was made to correlate the rate of amputation with the presence of open, comminuted fractures, but the findings are not significant. As may be expected, however, there was a direct correlation between rate of amputation and size of the wound.

Complications were encountered much less frequently than was expected. Hemorrhage from the suture line was quite rare. Infection at the repair site, which may result in hemorrhage or thrombosis, was rarely a problem. Latent thrombosis probably occurs more frequently than is realized; but the thrombus is often slow in forming, which allows time for the collateral circulation to increase and compensate for the major artery thereby preventing limb loss.

One of the greatest determining factors in the final results of arterial repair is the time lapse from arterial injury to repair. Even this period can be quite variable depending on many factors such as size of the wound, number of collaterals involved, level of the artery involved, ambient temperature, severity of shock and anatomic variations. Although it has been shown that results are proportionally

better when arterial repair is done within 10 hours after injury, an extremity may undergo irreversible muscle changes much earlier or remain viable much longer, depending on the factors mentioned. The author previously reported five cases with major arterial injury repaired from 11 to 24 hours after injury (an average of 16 hours). At the time of amputation, all five extremities exhibited good blood flow through necrotic muscles. It was considered that time-lag from injury to operation was a significant factor in those five patients.

The time from injury to repair in the 26 patients whose extremities subsequently required amputation varied from 1 to 24 hours, with a mean of 10.5 hours, which was almost identical with the average time-lag of 9.8 hours recorded for the entire series.

Numerous patients were seen with the injured limb cold, ischemic, anesthetic, paralytic, and with the joints fixed. As these limbs became warm and sensation and motion returned after arterial repair, they often began to swell and required a fasciotomy. When fasciotomy was delayed, all degrees of muscle necrosis occurred, varying from microscopic areas of focal necrosis to loss of complete compartments. The flexor compartment of the forearm and the anterior tibial compartment of the leg seemed to be most vulnerable.

Short-Term Follow-up

If analogous groups of cases are compared after follow-up studies limited to the theater, findings reported by Ziperman showed that there were 127 major arterial repairs in 1952, with 26 cases (20.5 per cent) resulting in amputation. In this study for 1953, of the 193 major arterial repairs with limited follow-up studies there were 26 cases (13.5 per cent) which resulted in amputation, an improvement of 34.1 per cent in saving of limbs during the last year of the Korean conflict.

Thrombosis was reported as occurring in 29 cases (17.4 per cent) of the total surviving unamputated patients with limited studies (Table 5). No arteriograms were made during these short-term studies. Thrombosis was considered as occurring when no pulse returned following arterial repair or when a pulse had been present postoperatively and later disappeared. Failure of a pulse to return following operation may have been the result of faulty technic rather than thrombosis, but the consequences are the same. Excluding the patients who underwent an amputation or died, thrombosis occurred in 15.2 per cent of the vessels repaired by direct anastomosis, in 30.0 per cent of those repaired by autogenous vein grafts, and in 62.5 per cent of those repaired by homologous arterial grafts. Lateral repair resulted in only 5.6 per cent thrombosis.

Late Follow-up Studies

Since 62 of the 193 repairs in surviving patients could not be followed, only 131 cases in which there were late follow-up studies are

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Table 5. Short-Term Follow-up on Major Vascular Repairs in Korea, 1953*

Repair	Total Num- ber	No Amputation				Amputated		Number of Deaths
		Good		Thrombosed		Num- ber	Per Cent	
		Num- ber	Per Cent	Num- ber	Per Cent			
Anastomosis-----	136	95	84. 8	17	15. 2	13	10. 4	11
Vein graft-----	30	14	70. 0	6	30. 0	8	28. 6	2
Arterial graft-----	12	3	37. 5	5	62. 5	3	27. 3	1
Lateral repair-----	20	17	94. 4	1	5. 6	1	5. 3	1
Conservative-----	4	4		0		0		0
Release spasm-----	2	1		0		1		0
Remove thrombus-----	1	1		0		0		0
Ligation-----	6	3		0		0		3
Total-----	211	138		29		26		18
Average-----			82. 6		17. 4		13. 5	

*Percentages exclude cases which underwent amputation or died.

included (Table 6). Counting the same 26 amputations reported in the short-term group which remain unchanged for this latter group of 131, the amputation rate is increased to 19.8 per cent.

Table 6. Late Follow-up on Major Vascular Repairs in Korea, 1953*

Repair	Total Num- ber	No Amputation				Amputated		Number of Deaths
		Good		Thrombosed		Num- ber	Per Cent	
		Num- ber	Per Cent	Num- ber	Per Cent			
Anastomosis	96	62	86. 1	10	13. 9	13	15. 3	11
Vein graft	24	9	64. 3	5	35. 7	8	36. 4	2
Arterial graft	11	3	42. 9	4	57. 1	3	30. 0	1
Lateral repair	8	6	100. 0	0	0	1	14. 3	1
Conservative	3	3		0		0		0
Release spasm	2	1		0		1		0
Remove thrombus	1	1		0		0		0
Ligation	4	1		0		0		3
Total Average	149	86	82. 0	19	18. 0	26	19. 8	18

*Percentages exclude cases which underwent amputation or died.

An attempt to determine the exact number of thromboses was difficult even in this late group, because it was not possible to do an arteriogram on every patient. The volume of the pulse distal to the repair site is not always indicative of the condition of the repair. At some levels, collateral vessels are quite adequate or develop quite rapidly. Even though some patients with a poor pulse have been shown by arteriogram to have a patent but constricted repair, all patients with a poor pulse or absence of pulse following operation have been recorded in this study as thrombosed.

Thrombosis was reported as occurring in 19 (18 per cent) of the 105 surviving, unamputated patients on whom there were late follow-up studies (Table 6). Of the unamputated patients whose vessels were repaired by anastomosis, 13.9 per cent thrombosed. Excluding the case amputated, none with lateral repair were reported as thrombosed. In the 21 patients whose vessels were repaired by grafts, 14 autogenous vein grafts and 7 homologous arterial grafts were used. In this group, 35.7 per cent of the vein grafts thrombosed, as compared to 57.1 per cent of the arterial grafts. From previous experience with homologous arterial grafts, this percentage is not surprising; it was not expected, however, that it would be as high with the autogenous vein grafts.

Discussion

Admittedly, the Korean conflict offered many advantages over World War II in regard to vascular repair. During the latter phase of the conflict, the front line in Korea was relatively stable. The surgical hospitals were within 6 to 12 miles of the front and there was ample air cover, plus the advantage of more rapid evacuation by helicopter. The expanded antibiotic armamentarium and availability of new vascular clamps, plus the experience gained in vascular surgery since World War II, all contributed to the success of vascular surgery in the Korean conflict. The Potts ductus and coarctation clamps contributed immensely to the success of the entire vascular surgery program.

Obviously, once an amputation was reported, the late follow-up concerning that patient's extremity was known. By far the highest percentage of amputations occurred at the initial installation; thus they were reported. While there were 26 amputations (20 per cent) reported in 131 patients in the late follow-up group, this percentage is believed to be too high. If all of the 62 patients whose cases were not followed had viable limbs, then the amputation rate was 13.5 per cent. This rate was certain to be low because amputations were recorded outside of the Korean theater. The actual amputation rate for the entire series probably ranged between 13.5 per cent and 19.8 per cent, and possibly near the mean of approximately 16.5 per cent.

In order to present an accurate picture of the rate of thrombosis, findings were included on both the entire group of 193 patients with limited studies and on the late follow-up group of 131 patients. Ten thromboses are known to have existed in the 62 patients lost to follow-up. The number of thromboses in the amputated patients is unknown; hence the rate of thrombosis was figured for only the living unamputated patients. When rates of thrombosis of the short-term follow-up group and the late follow-up group were compared, they were found to be almost identical (Tables 5 and 6). If these findings are any criteria for the most satisfactory methods of repair, then the preferred methods, in order, are: lateral repair, direct anastomosis, autogenous vein graft, and homologous arterial graft. However, it must be borne in mind that lateral repair was used for only the simplest lacerations, while the more extensive wounds required a more complicated repair.

Lateral repair should be reserved for only minor, clean-cut lacerations of an artery. Large irregular lacerations are better excised with repair by direct anastomosis. By the same token, if the damaged area of the vessel is large, it is often wiser to débride thoroughly and insert a graft, rather than to sacrifice important collaterals in order to perform an anastomosis. An anastomosis under undue tension tends to separate or to create spasm with resulting thrombosis.

Even though a number of limbs had to be amputated and others were crippled by loss of muscle tissue or had complications of nerve injury or open fractures, there was a significant saving of limbs in Korea by repair of acute vascular injuries.

Summary

1. An attempt was made in Korea during 1953 to carry out follow-up studies on every soldier having a major artery repaired. These findings were compared with findings reported from Korea during 1952.

2. During 1953 there were 211 major arterial repairs in 205 patients reported in Korea. Death occurred in 15 patients with 18 repairs. Of the surviving patients, 26 required limb amputation.

3. Of the 193 repairs in surviving patients with short-term follow-up studies during 1953, amputation resulted in 13.5 per cent of the cases. This is an improvement of 34.1 per cent over an analogous series of 127 cases with 20.5 per cent amputations reported for 1952.

4. Sixty-two of the 193 patients were lost to follow-up studies, leaving 131 with the late follow-up studies of which 26 required amputation (19.8 per cent).

5. The correct amputation rate for the entire series ranged between 13.5 per cent and 19.8 per cent, or possibly near the mean of approximately 16.5 per cent.

6. A comparison of the rates of thrombosis in repairs with short-term follow-up studies and those with late follow-up studies showed the findings to be almost identical. If absence of thrombosis is an indication of superior methods of repair, then in order of preference the methods are: lateral repair, direct anastomosis, autogenous vein graft, and homologous arterial graft. The results of the study indicated a close correlation with the severity of the vascular wound.

References

1. De Bakey, M. E., and Simeone, F. A.: Battle Injuries of the Arteries in World War II. *Ann. Surg.* 123: 534, 1946.
2. Seeley, S. F.; Hughes, C. W.; Cooke, F. N., and Elkin, D. C.: Traumatic Arteriovenous Fistulas and Aneurysms in War Wounded. *Am. J. Surg.* 83: 471, 1952.
3. Cooke, F. N.; Hughes, C. W.; Jahnke, E. J., Jr.; and Seeley, S. F.: Homologous Arterial Grafts and Autogenous Vein Grafts Employed in Order to Bridge Large Arterial Defects in Man. *Surgery*, 33: 183, 1953.
4. Seeley, S. F.; Hughes, C. W.; and Jahnke, E. J., Jr.: Direct Anastomosis Versus Ligation and Excision in Traumatic Arteriovenous Fistulas and Aneurysms. *Surgical Forum*, Clinical Congress of the American College of Surgeons, 1952, pp. 152-154. W. B. Saunders Company, Philadelphia, 1953.
5. Hughes, C. W.: Acute Vascular Trauma in Korean War Casualties. *Surg., Gynec. & Obst.* 99: 91, 1954.
6. Jahnke, E. J., Jr., and Seeley, S. F.: Acute Vascular Injuries in the Korean War. *Ann. Surg.* 138: 158, 1953.
7. Jahnke, E. J., Jr., and Howard, J. M.: Primary Repair of Major Arterial Injuries. *Arch. Surg.* 66: 646, 1953.
8. Ziperman, H. H.: Acute Arterial Injuries in the Korean War. *Ann. Surg.* 139: 1, 1954.

Chapter 11

Acute Vascular Trauma in Korean War Casualties:

An Analysis of 180 Cases*

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Acute injuries of major arteries always constitute a threat to life and limb. During time of war, these injuries assume even greater importance because of the epidemic increase in numbers of such lesions. The Korean conflict provided the opportunity for the observations recorded in this communication. This analysis, which embodies 79 injuries of major arteries, 30 of minor arteries, and 71 of major veins, comprises the personal studies of one of three observers working independently in Korea as members of the Surgical Research Team of the Army Medical Service Graduate School, Washington, D. C. Findings of the other two observers have already been reported.^{1,2} At the same time, a similar program was under way by Navy medical officers in the Marine hospitals in Korea.³

The most complete review of acute vascular injuries is the De Bakey and Simeone analysis of 2,471 cases from World War II.⁴ They found that the incidence of amputations from injury to major vessels of the extremities was 49.6 per cent. Of 81 suture repairs of these lesions, the amputation rate was 35.8 per cent. In the initial phase of the Korean conflict, attempts at anastomosis of damaged arteries were only slightly successful.⁵

Major Artery Injuries

During a 6-month period in Korea, 79 consecutive injuries to major arteries in 76 unselected patients were treated by the author at the 8055th Mobile Army Surgical Hospital (later named the 43d Surgical Hospital). The distribution of these injuries is presented in Table 1.

The 79 vascular injuries encountered were broken down as follows: lacerated 42, severed 31, contused with spasm 2, and contused with thrombosis 4. Most patients had multiple injuries. Complicating these were severe open, comminuted fractures present at the site of the arterial injury in 12 cases, with less severe open fractures present in 5 other cases (Fig. 1). All but 10 wounds (12.7 per cent) were caused by fragmenting missiles.

*Previously published in *Surgery, Gynec. & Obst.* 99: 91, 1954.

Table 1. The Distribution of the Major Arterial Injuries

Vessel	Number of Cases
Common carotid	4
Axillary	3
Brachial	25
External iliac	1
Common femoral	3
Superficial femoral	22
Popliteal	21
Total	79

Thirty of the patients arrived in shock of varying severity. Thirty-seven were admitted with a tourniquet in place which had been applied from 40 minutes to 14.5 hours previously, the average time being 4 hours. The over-all time from injury to surgery averaged 10 hours with extremes from 3.5 to 28.5 hours. Of this interval, evacuation time to the hospital averaged 5.8 hours and time from admission to surgery an additional 4.2 hours. Resuscitation was often a problem and, although the patients were sometimes admitted soon after injury, the time lost during resuscitation sometimes militated against recovery of the limb. This is a reflection of the severity of injury and shock in these patients.

Concomitant nerve injury, not necessarily at the site of the vascular injury, was evident in 36 of the 79 vascular injuries. Of these, there were 20 major nerve injuries, 9 minor nerve injuries, and 7 instances of nerve involvement believed due to anoxia, tourniquet, or mild contusion. Because of the multiplicity of wounds, prolonged use of the



FIGURE 1. Successful vascular repairs were performed in the presence of such open, comminuted fractures.

tourniquet, and periods of anoxia, it was difficult to determine the cause of nerve injury in some cases when the actual damage was not visualized.

Methods

Major vascular injuries were given priority evacuation along with other priority cases. High priority is important for resuscitation and surgery since every minute may be vital in saving a limb.

Preoperative oscillometric studies were made on limbs showing absence of a pulse, unless the limb was in traction or a tourniquet was in place. Twenty-two of the 32 patients admitted without tourniquet had no palpable pulse in the injured extremities. Oscillometric readings in these limbs were almost universally zero, with only three showing a quiver of the oscillometer needle. Immediately following arterial repair, some cases showed better oscillometric readings on the repaired side than on the normal side but the group as a whole averaged 0.67 unit less on the repaired side.

An anatomic surgical approach was used regardless of location of the wound. An adequate incision was always made and proximal control of the vessel secured first. All repairs were by suture with 00000-braided arterial silk, continuous everting mattress technic, intima to intima placement.

Two Pott's ductus or coarctation clamps plus arterial tissue forceps, a fine needle holder, and 00000-braided arterial silk were the only additional equipment found necessary for peripheral vascular surgery. Each hospital receiving such patients should be so equipped.

All wounds were adequately débrided, but all collateral vessels were preserved where possible. Even after arterial reconstruction, it often took the damaged muscle some time to bleed, making it difficult to evaluate properly the viability of the muscle distal to sites of arterial injury.

The repaired vessels were covered for nourishment and to avoid secondary infection. Coverage should be loose but adequate, and without primary closure of the skin. If infection occurs, it will often result in thrombosis at the site of repair.

To be sure there was no undue tension on the suture line, repairs at joint sites were inspected at completion of surgery by fully extending the joint and visualizing the repair site. These limbs were then flexed 10 to 15 degrees for 2 weeks, being restrained only with adhesive tape to allow flexion but to limit extension. Limited active motion to improve the circulation was encouraged as soon as possible after surgery. When active motion was not possible, passive motion was utilized.

Delayed closure of clean wounds was carried out on the fourth to sixth postoperative day when the patient work load permitted. If

wounds remained clean and healing uneventful, joints were gradually, fully extended and the patient ambulated after 2 weeks.

When bed space permitted, these patients were observed at the surgical hospital for 8 to 10 days, then evacuated to Army hospitals in Japan where primary follow-up studies were initiated. Those eligible for evacuation to the United States were subsequently evaluated further by arteriography, oscillometry, and skin temperature studies at Walter Reed Army Hospital. Inasmuch as some of the casualties were Ethiopian, Korean, Colombian, and Commonwealth patients, long-term follow-up studies were possible only on the Americans.

It is fully realized that long-term follow-up studies will be necessary for a complete evaluation of acute arterial repairs. However, it appears at this time that a very high percentage of limbs with arterial damage can be saved if the patient can be evacuated to a hospital and resuscitated before irreversible muscle changes take place.

Results

Six patients with 7 arterial repairs (popliteal 4, superficial femoral 2, common femoral 1) died shortly after operation from causes other than arterial injury. For this reason, adequate evaluation was not possible and these cases are eliminated from the analysis, except to mention any complication occurring before death. Summaries of these are given in the addendum. Of the 72 remaining patients who survived, 8 subsequently came to amputation of the involved limb. Thus, 11.1 per cent of the limbs were lost and 88.9 per cent were saved.

The indications for the 8 amputations warrant some explanation. Five of these were performed within the first postoperative week and three subsequently. Seven were in lower extremities and one in an upper extremity. Five amputations resulted from irreversible muscle changes which probably had taken place prior to vascular repair. These muscle changes were obvious at the time of reparative surgery in three cases, and the viability of the muscles was questionable in the two others. Muscle biopsies at the time of repair confirmed the gross observations. Cold injury was a complication in two of the amputees. In these five cases, the time from injury to repair varied between 11 and 24 hours, with a mean of 16 hours. Restoration of blood flow was effected in the hope of saving the limb or, at least, providing the longest possible viable stump. At the time of amputation there was a brisk flow of blood in the already devitalized muscles. If these five cases with irreversible muscle damage at operation are excluded, repair in this series was successful 95.8 per cent and amputation was required in 4.2 per cent.

A sixth patient required amputation because of venostasis, with ensuing liquefaction of the muscles. The seventh amputation resulted from slippage of the fractured femur with compression and thrombosis of the repaired artery. The eighth patient required a transmetatarsal amputation 3 months later for dry gangrene of the toes; probably the result of a small embolus from the repair site and not directly due to initial failure of the repair.

Table 2. Resulting Amputations, Vessels Injured, and Causes

Vessel	Injury	Injury to Repair (hours)	Type of Repair	Cause of Amputation
Axillary	severed	24	anastomosis	muscles had undergone irreversible changes prior to vascular repair; good blood flow was present through dead muscle at amputation
C. femoral	spasm	11	release of spasm	
C. femoral	laceration	14.5	arterial graft	
Popliteal	laceration	16.5	anastomosis	
Popliteal	severed	13.75	arterial graft	
Popliteal	laceration	11	anastomosis	venostasis
Popliteal	severed & lacerated	6.5	vein graft	compressed by slipping of open, com. frac. femur
Popliteal	severed	12	anastomosis	partial amp. foot 3 months later, probably due to thrombosis

Of the repairs in limbs subsequently amputated (Table 2), grafts were employed in 37 per cent of the cases, as compared to 19 per cent for the whole series. This is a further indication of the extent of the trauma in these patients. It should also be noted that the injuries were to arteries at critical levels which may be expected to result in gangrene in a high percentage of cases if ligated.

Factors Influencing Saving of Limb Following Blood Vessel Surgery

Time-Lag

It is not possible to establish a specific time interval after which repair is no longer practicable because of muscle necrosis. There are many variables which make it impossible to determine clinically when muscle changes are irreversible. Factors to be considered are the length of time after injury, severity of the wound, the artery injured, the level of the injury, the number of collaterals damaged, the outside temperature, exposure, the state of shock, and anatomic variations. A decrease in the surrounding temperature (short of causing frost-

bite) appears to be advantageous. The converse also seems to be true. During very hot weather, muscle changes and gangrene have been observed to occur much more rapidly.

Acute vascular injuries should be repaired in borderline cases of irreversible muscle damage, even though the limb is expected to be lost. Restoration of blood flow may preserve some length of the limb or even a joint before a line of demarcation appears.

Tourniquets

The application of a tight pressure dressing was not always adequate for control of hemorrhage as evidenced by 47 per cent of these patients admitted with a tourniquet in place. Without control of the hemorrhage, resuscitation cannot be accomplished. Prolonged use of the tourniquet for resuscitation may result in a nonviable extremity, so a vicious cycle is created. In such cases, the bleeding artery was exposed and a clamp was applied to the very end of it in order to preserve all of the artery possible for repair. A pressure dressing was then usually adequate, and resuscitation could be continued.

Many physicians are still not tourniquet-conscious. Tight Esmarch tourniquets have been found covered by external dressings. It is believed that a tourniquet should be applied in the field and left in place until removed by a medical officer at a time when blood is available to resuscitate the patient. A pressure dressing will usually suffice if it is applied tight enough to control the hemorrhage. If watched carefully, the pressure dressing can be loosened after a matter of 20 to 30 minutes, but continued observation is necessary to prevent recurrence of hemorrhage as blood pressure rises.

Muscle Changes

Many patients were admitted with muscle hardened by ischemia with contractures and fixed, frozen joints. These extremities were cold, spastic, anesthetic and paralytic. After restoration of blood supply, the muscles swelled and fasciotomies were performed. At fasciotomy, the muscle was often found to be pale, swollen, and ischemic, and a blood-tinged transudate often ran out as the muscle bulged from the compartment. It was observed that muscles of the anterior tibial compartment of the leg and the flexor compartment of the forearm were the most vulnerable to swelling and broke down more quickly than others if fasciotomy was not done early. As more experience was gained, fasciotomy was performed prophylactically in borderline cases.

After restoration of blood supply and the initial swelling, such extremities became warm; the swollen muscles softened, relaxed, and sensory and motor function gradually returned. This process sometimes took 2 to 3 days. In the lower extremities, peroneal nerve function was invariably slow to return.

Serial muscle biopsies were taken in many cases showing various stages of degeneration, fragmentation, hyalinization and regeneration of muscle. In other instances, microscopically normal muscle was seen after prolonged periods of anoxia. This project was undertaken to study the condition of muscle and its blood supply at the time of surgery and to observe the progression of muscle degeneration and regeneration. It was hoped that the cause of irreversible muscle damage might be delineated as being due to muscle fiber death or secondary to venous, capillary, and arterial thrombosis.

In some of the above cases, changes developed varying from microscopic areas of focal necrosis to loss of whole muscle compartments. Some of these muscles underwent complete sterile liquefaction. On dissection of these extremities after amputation, many small veins were found thrombosed while the arterioles were patent. One of these patients came to amputation 18 days after repair because of slow muscle changes. One compartment after another was débrided until only skin, bone, nerve and vessels remained.

It is possible in some cases that restoration of the blood flow hastened muscle degeneration. Experimental work has shown that rapid revascularization of ischemic muscle hastens degeneration.⁶ It has been suggested that there may be some similarity to frostbite where a slow, rather than rapid, return of blood flow and warmth may be important to survival of damaged muscle.⁷

In the lower extremity, when muscle degeneration had taken place before restoration of blood supply, it was invariably found at amputation that the soleus muscle had undergone more extensive degeneration than the gastrocnemius.

These muscle studies are still in progress and will be reported separately and in detail at a later date.

Types of Repair

While simple suture repairs of lacerated arteries were held to a minimum in this series, some were done by others in the Theater. Except for the small, clean, knife-like laceration, it is difficult to evaluate, débride and suture the damaged artery without constriction of the lumen. It is usually better to exercise the damaged portion and do an end-to-end anastomosis.

It was possible to do a direct anastomosis in 65 per cent of the cases (Table 3). The portion of artery excised averaged 0.75 inch. With dissection of the artery for approximately 2 inches in each direction, an anastomosis could usually be done without undue tension (Figs. 2 and 3). Major branches and collaterals were preserved, although minor branches were at times sacrificed to gain arterial length.

Table 3. The Frequency and Types of Repair Utilized

Type of Repair	Number of Cases
Primary anastomosis	51
Autogenous vein graft	11
Homologous arterial graft	4
Suture repair	3
Removal of thrombus	1
Release of spasm	2
Conservative treatment	4
Ligation	3
Total	79

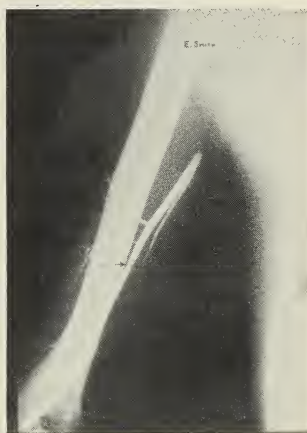
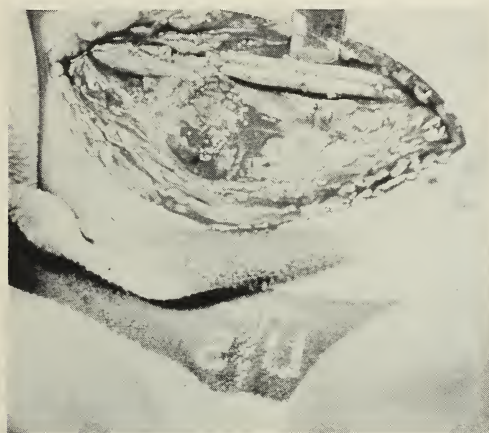


FIGURE 2. A brachial artery anastomosis and arteriogram showing the repair site several months later.



FIGURE 3. A popliteal artery anastomosis and arteriogram showing the repair site several months later.

Excised sections of damaged arteries were studied histologically for adequacy of débridement. Several sections taken from the cut ends of the artery adjacent to the anastomosis have shown microscopic changes including polymorphonuclear infiltration and irregularities of the internal elastic membrane, which might be interpreted by some as being due to inadequate débridement. Follow-up studies have shown no correlation with postoperative thrombosis in these cases.

Some anastomosed segments removed from amputated limbs and at autopsy have shown microscopic areas of infarction at the suture line. It has not been determined whether these changes represent inadequate débridement or changes secondary to the stripping of adventitia and vasa vasorum incident to suture of the vessel. It is possible that such infarctions may occur in many cases and that the infarcted area is replaced in the same manner as a graft. Details of histologic studies of arteries will be reported in a separate communication.

Grafts were required in 19 per cent of the cases (Table 3). Arterial defects up to 4.5 inches in length were encountered but, as already mentioned, the defects averaged 0.75 of an inch. Both autogenous vein grafts and homologous arterial bank grafts were utilized. Autogenous vein grafts from 0.75 inch to 3.75 inches and homologous artery grafts from 1 inch to 4.25 inches were used (Fig. 4). The saphenous vein was most often used as a graft; however, the cephalic, basilic and others were also used. The saphenous vein is usually of adequate size for most peripheral vessels and appears to be stronger than veins of upper extremities. Vein grafts are always turned end for end, when inserted, so the valves do not obstruct blood flow. Grafts usually are not taken from the involved limb, especially if other major veins of the limb are damaged. Sometimes the companion vein of the artery may be utilized, if already damaged, but usually it is too large. Vein grafts should be chosen slightly smaller than the artery into which they are to be inserted to prevent overdilatation.

Homologous arterial bank grafts were preserved in a modified Tyrode's nutrient graft solution, up to 30 days.⁸ Availability of bank grafts saves time and is advantageous in cases in which an autogenous vein graft is not always readily accessible because of the patient's position at surgery.

Autogenous vein grafts have been more satisfactory than homologous arterial grafts. This has borne out our previous experience.⁹ Although no limbs were lost as a result of it, two of the four homologous arterial grafts in this group thrombosed within 4 to 8 weeks. The other two, utilized in extremities which came to amputation, were patent at the time of amputation 6 days after insertion.

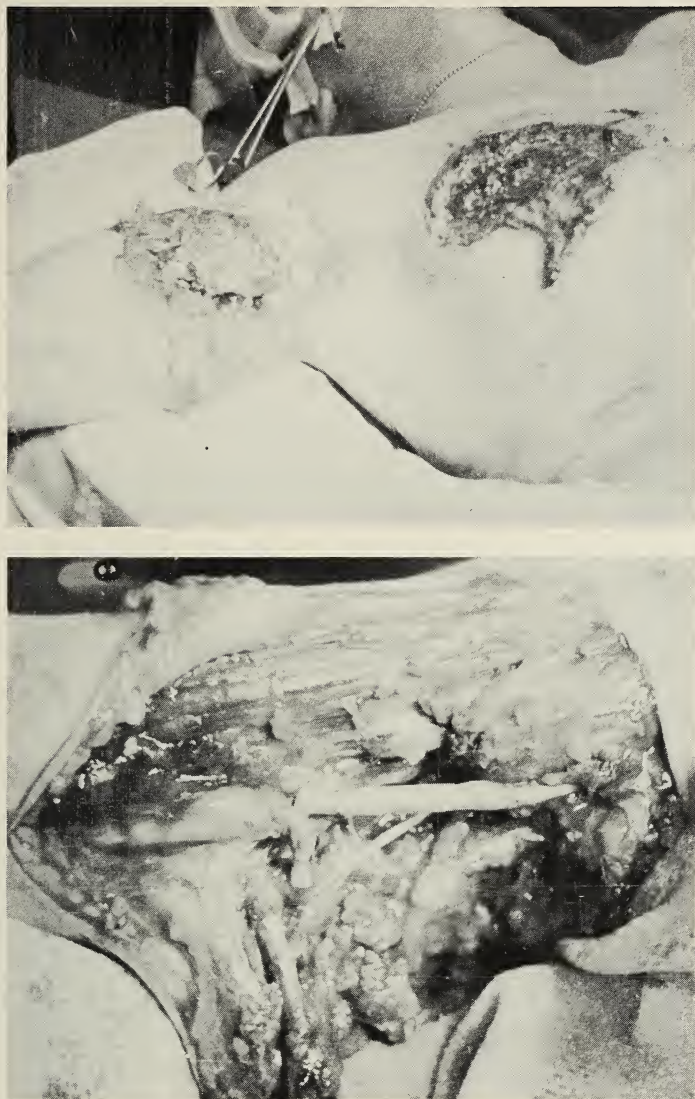


FIGURE 4. An example of a massive avulsing wound of the upper arm prior to surgery and after debridement and insertion of a 3-inch saphenous vein graft into the brachial artery.

Ligation was reserved for noncritical arteries in which it was deemed unwise to do a repair, and for patients who could not tolerate prolonged surgery. Of the 3 major arteries ligated in this series, the terminal brachial, involving ulnar and radial bifurcation, and the terminal popliteal, below the origin of the anterior tibial artery, were involved. Two of these patients were in such poor condition that they could not tolerate further surgery. In each case it was determined at surgery that the limb had adequate circulation for viability.

Ligation of the concomitant vein at the time of artery ligation was not practiced and it is believed that it may even be a harmful procedure.

Previously, a very high percentage of injuries of the carotid arteries, ligated or repaired immediately, resulted in death or hemiplegia. It has been found in partially severed major arteries of extremities that, if ligation is delayed several hours or days, a much lower incidence of gangrene occurs. Such arteries may be ligated weeks later and gangrene is unusual. On this basis, it was decided that when the hematoma was self-contained and the hemorrhage controlled, all carotid injuries would be treated conservatively, particularly if an arteriovenous fistula was present. The wounds were débrided superficially and great care was taken not to dislodge the clot and to avoid wound infection. It was believed that even if the patient bled 1 or 2 days later, his chances of survival were much better after delayed rather than immediate surgery. There has been no occasion to regret this decision. Of the four cases in this group treated conservatively, three have since had successful definitive surgery and the fourth apparently had spontaneous closure of the arterial defect.

The same conservative treatment was applied to self-contained, high subclavian injuries because of the difficult surgical approach, long operation, and dangers of massive hemorrhage and infection. Such lesions were allowed to develop an arteriovenous fistula or false aneurysm and then were operated on subsequently.¹⁰

While follow-up studies sometimes show some constriction at the suture line, actual thrombosis has been quite rare. These studies have also shown the repaired limb to average 1.6 oscillometric units less than the normal side. Several factors may influence these oscillometric changes—such as the multiplicity of wounds, nerve injury, scar tissue, or any constriction present at the repair site. These same changes may also alter skin temperature changes which have averaged one degree less in the limb with the repaired vessel in the follow-up studies.

Complications

There must be close observation for postoperative complications of infections and hemorrhage. Infection may result in thrombosis or dehiscence of the arterial suture line with hemorrhage. Penicillin and streptomycin were used prophylactically in these cases and usually the infection was not a problem. In fact, only one severe infection occurred; this was after a vein graft and resulted in thrombosis of the graft. Dehiscence and hemorrhage may also occur from a poor anastomosis, inadequate arterial débridement, inadequate arterial coverage, or too much tension.

Hemorrhage from the suture line in the immediate postoperative period was not observed. It was reported in three cases in follow-up studies. One such patient who was anuric, and who was dialyzed, had repeated convulsions and bled from his suture line into a nonhealing wound 12 days following operation. This patient died. A second patient bled from his wound following delayed skin closure 21 days after operation. The peripheral pulses were unaltered. After a second hemorrhage 36 days postanastomosis, the pulses gradually disappeared. There was no surgical intervention or proof of the site of the hemorrhage, but this patient had a severe open, comminuted fracture of the femur and had been in traction during this period. The artery may easily have been lacerated by a bone fragment. This patient was still in traction 9 months later. A third patient bled from his suture line 14 days postoperatively. The vessel was ligated without limb loss. Two other patients had hemorrhage as a complication but not from the suture line. One of these, the third fatality listed in this series, had an open, comminuted fracture of the femur which lacerated the artery above the repair site. The other case of hemorrhage occurred 11 days following injury with hemorrhage from the artery one inch above the anastomosis, presumably from a damaged area in the artery, overlooked at the time of surgery; this necrosed later.

Thrombosis was not a problem in arterial repair. Anticoagulants were not used and are not considered necessary.

Although amputation in one case resulted from arterial spasm alone (*see* Table 2), this was rarely a problem. Spasm was treated variously at the time of surgery with 2.5 per cent papaverine hydrochloride, warm saline packs, segmental periarterial sympathectomy, and 25 to 50 mg. of intra-arterial priscoline. Postoperatively, repeated sympathetic blocks with 1 per cent procaine were used as indicated. Sympathectomy was not done after repair of these acute injuries. A second patient, treated for severe segmental spasm only, still has a patent artery but a slight constriction throughout the length of the site of the original spasm. It is not known whether this is due to changes in the arterial wall, a thrombus within the artery, or to scar tissue and callus formation from the associated open fracture.

The trauma causing the arterial damage, the vasoconstriction of the collaterals, plus shock and its accompanying peripheral vasoconstriction, all combine to decrease the blood flow to a limb with arterial injury. As a result of this chain of events, some patients having major arterial damage, even for a short period of time, may exhibit muscle changes resulting from anoxia.

Open, comminuted fractures did not cause as high a percentage of complications as expected. While severe open, comminuted fractures occurred in only 15 per cent of the arterial injuries, direct complica-

tions could only be attributed to the fractures in two cases previously mentioned. In one, the fracture slipped, compressing the repaired artery and requiring amputation. In the other, laceration of the repaired artery was produced by the bone fragments, requiring ligation of the artery.

Such fractures complicate vascular repairs by slipping and compressing or lacerating the vessel. Tissue pulled between the artery and bone is protective. To avoid damage to the repaired artery, pin fixation in a cast may be necessary in cases such as fractures of the distal femur. Fixation in casts should be reserved for those with concomitant fractures. Such casts should be bivalved immediately and taped together to allow immediate access to the repair site in the event of hemorrhage.

Minor Artery Injuries

Thirty injuries to minor arteries were studied in other patients. Minor arteries injured included the profunda brachii, profunda femoral, radial, ulnar, anterior and posterior tibial. When only one of two minor arteries to an extremity was involved (such as an injured radial but intact ulnar), the injured vessel was ligated. In no case did this procedure lead to complication. In instances in which both vessels were involved, the limb was invariably so macerated and shattered that amputation was necessary. In such cases, one or both of the arteries would have been repaired had there been viability.

Vein Injuries

Concomitant major vein injuries were found in 50 cases with major arterial injuries. Other major vein injuries were added to this group to bring the total to 71. Fifty-eight of these veins were ligated. Complications ranged from none to various degrees of venostasis. In one patient, loss of an extremity resulted from severe venostasis. The popliteal, greater and lesser saphenous veins had been severed. While there was good arterial inflow, the muscles gradually liquefied and required amputation. On dissection of the leg, the arteries were all patent but the veins were all thrombosed. This observation has also been recognized by other investigators;³ and is similar to a massive venous thrombosis.

After complications were observed following venous ligation, major vein injuries, especially those at critical levels, were carefully evaluated for repair. An evaluation of an extremity should be made at the time of surgery to determine the extent of other vein injuries before a repair is performed. While the author has performed numerous venous repairs at elective surgery,¹⁰ it was realized there was

more danger of thrombosis and embolism in acute trauma. However, venous repair is believed to be a safe procedure if the clots are carefully removed and the vein is properly débrided and sutured. The size and number of wounds, size and level of vein injured, and the number of collateral veins injured are all factors to be considered in determining whether or not a vein should be repaired.

Venous repairs were performed by simple over-and-over suture except for one case in which anastomosis was carried out by a continuous everting type suture. Only 13 major veins were repaired in this group, 12 by lateral suture and 1 by end-to-end anastomosis (Table 4). No anticoagulants were used in these venous repairs.

Table 4. Locations of Major Veins Repaired

Vessel	Number of Cases
Popliteal	5
Common femoral	3
Vena cava	1
External iliac	2
Superficial femoral	2
Total	13

Two patients early developed a thrombophlebitis of the superficial veins of the leg, but in each case the greater saphenous system had been previously ligated at the time of surgery. Follow-up studies have shown that when repair resulted in decrease in size of the lumen, a high percentage of the veins thrombosed. Thrombosis occurred after the initial 8- to 10-day period of observation. Venostasis has not been a major problem at follow-up. It is difficult to place the cause of persistent swelling in some cases because many have neurologic, venous, lymphatic, and multiple muscular injuries.

Summary

1. One hundred eighty cases of acute vascular injuries to soldiers wounded in the Korean conflict were studied. These consisted of 79 major artery injuries, 30 minor artery injuries, and 71 major vein injuries.

2. After repairs of the major arterial injuries, 88.9 per cent of the limbs were saved and 11.1 per cent amputated.

3. The amputations included five limbs which had undergone irreversible muscle changes prior to vascular repair. The other three amputations resulted from complications of repairs.

4. Fasciotomies were found to be valuable adjuncts after arterial repair in limbs with borderline muscle changes.

5. A specific time interval after which arterial repair could not be performed was not established.

6. Tourniquets should be applied and left in place until they can be removed by a doctor who has adequate blood available for resuscitation.

7. All degrees of muscle degeneration to regeneration were observed in cases of delayed arterial repair.

8. The major methods of arterial repair were end-to-end anastomosis and use of autogenous vein grafts and homologous artery grafts. Autogenous vein grafts in arteries have been far more satisfactory than homologous artery grafts.

9. Complications were rare, and included postoperative hemorrhage, infection, spasm and thrombosis. Antibiotics were used. No anticoagulants were administered.

10. Thirty minor arteries were ligated without complications.

11. Seventy-one injuries to major veins were treated with some complications which led to the successful repair of 13 major veins.

12. Immediate repair of injuries to major arteries is practical. All major arteries should be repaired except when the additional time required for surgery endangers the patient's life. While some limbs survive major artery ligation, they are not functionally equal to the limb with a successful vascular repair. If thrombosis should occur at the repair site, the process will often be slow enough so that a collateral blood flow will develop and provide adequate circulation to the limb. If patients with vascular injuries can be brought to surgery before irreversible muscle changes take place, a very high percentage of limbs can be saved.

Addendum

1. The first death occurred in a patient who was admitted in deep shock with an injury of the common femoral artery. Cardiac arrest occurred soon after induction of anesthesia; and he was resuscitated by cardiac massage. While his chest was being closed, an artery graft was inserted into the defect of the femoral artery. He died 4 hours later.

2. A second patient had a severed popliteal artery plus injuries to all extremities, abdomen, and chest. He was in deep shock for many hours. The artery was anastomosed while abdominal surgery was being performed. Postoperatively, the patient lived only 38 hours.

3. A third patient with superficial femoral artery injury and multiple other injuries was admitted in severe shock. His condition was so poor that, after anastomosis of the artery, other débridements were not completed. He continued to be irrational after surgery and, although placed in a spica cast, the open fracture of the femur lacerated the artery above the repair site on the third postoperative day.

Since his condition did not permit prolonged surgery, the artery was ligated. The leg was amputated 9 days later. The patient died of cerebral damage a few days after evacuation.

4. The fourth patient was admitted in severe shock with a severed superficial femoral artery and cerebral anoxic changes. After a quick, easy arterial repair, the patient continued to be mentally hazy and became anuric. He was evacuated to the Renal Insufficiency Center. He had repeated convulsions, and 12 days after surgery he hemorrhaged from the arterial repair. The artery was ligated. Two days later the patient died of cardiac arrest during an attempted further débridement.

5. A fifth death occurred in a patient with a popliteal artery injury. The arterial injury was easily and successfully repaired but the patient died following a subsequent negative abdominal exploration at another hospital.

6. The sixth death occurred in a patient admitted in severe shock with extensive injuries to both popliteal arteries, buttocks, back, all extremities, and open, comminuted fractures of the femur. One popliteal artery was anastomosed while a colostomy was being carried out. The other popliteal artery was ligated in its terminal portion below origin of the anterior tibial artery. The patient's condition did not permit completion of his débridement. Later at a second operation, he developed pulmonary edema and died.

References

1. Jahnke, E. J., Jr., and Seeley, S. F.: Acute Vascular Injuries in the Korean War. *Ann. Surg.* 138: 158, 1953. (Chapter 8 of this volume.)
2. Jahnke, E. J., Jr., and Howard, J. M.: Primary Repair of Major Arterial Injuries. *Arch. Surg.* 66: 646, 1953.
3. Spencer, F.: Personal communication. (Johns Hopkins Hospital, Baltimore, Maryland.)
4. De Bakey, M. E., and Simeone, F. A.: Battle Injuries of the Arteries in World War II. *Ann. Surg.* 123: 534, 1946.
5. Hughes, C. W., and Bowers, W. F.: Observations on Blood Vessel Surgery. *Am. J. Surg.* 85: 174, 1953.
6. Harman, J. W., and Gwinn, R. P.: The Recovery of Skeletal Muscle Fibers from Acute Ischemia as Determined by Histologic and Chemical Methods. *Am. J. Path.* 25: 741, 1949.
7. Brooks, B.: Pathologic Changes in Muscle as a Result of Disturbances of Circulation. *Arch. Surg.* 5: 188, 1922.
8. Cooke, F. N.; Kuhns, D. M.; Elston, J. T.; Dozier, S. M., and Fusillo, M. H.: Blood Vessel Bank. *U. S. Armed Forces Med. J.* 2: 1779, 1951.
9. Cooke, F. N.; Hughes, C. W.; Jahnke, E. J., Jr., and Seeley, S. F.: Homologous Arterial Grafts and Autogenous Vein Grafts Employed in Order to Bridge Large Arterial Defects in Man. *Surgery* 33: 183, 1953.
10. Seeley, S. F.; Hughes, C. W.; Cooke, F. N., and Elkin, D. C.: Traumatic Arteriovenous Fistulas and Aneurysms in War Wounded. *Am. J. Surg.* 83: 471, 1952.

Chapter 12

The Pathology of Skeletal Muscle Ischemia in Man:

A Description of Early Changes in Extremity Muscles Following Damage to Major Peripheral Arteries on the Battlefield

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During time of war, the treatment of vascular trauma becomes a major problem for the surgeon. Most of the progress in this field has related to the perfection of technics for optional restoration of the interrupted blood flow. Relatively scant attention has been paid to the striking changes that take place in the tissues deprived of their nutrition. Since the changes occurring in devascularized skeletal muscle are of considerable significance to the surgeon who must decide whether or not to amputate, and what level of amputation to select, a pathologic study of ischemic skeletal muscle was undertaken in soldiers sustaining vascular trauma during and shortly after the Korean conflict.

Material and Methods

Muscle specimens taken from the extremities of 31 soldiers whose major peripheral arteries were acutely damaged form the basis of this report. The arterial injuries of 22 of the soldiers resulted from fragmenting missiles, those of 7 from bullets. One individual sustained vascular trauma in a vehicular accident. Another, who was not injured but developed spontaneous occlusion of a major extremity artery, is included in the series because of the similarity of the pathologic changes in his muscles to those of the trauma cases. The patients ranged in age from 19 to 40, with an average age of 24.

Skeletal muscle samples were obtained in three ways. (1) Biopsies (25 specimens from 19 patients) were taken during initial débridement of a wound, at the time of fasciotomy, or coincident with revision or closure of a wound. An effort was made to biopsy regions of muscle which had not been directly traumatized. Thus, the changes in most of the specimens were attributable only to deprivation of blood supply. Most of the biopsy samples were placed in saline for a period of 15 to 20 minutes (to prevent the appearance of certain artefactual changes) and were fixed subsequently in a 10 per cent formalin solu-

tion. (2) Surgical excision of degenerating muscle groups (3 cases). (3) Amputation (16 cases). In most instances the entire amputated specimen was received by the pathologist. In a few cases the specimen was dissected by the surgeon, and formalin-fixed samples of various muscles were forwarded to the laboratory.

The clinical aspects of the patients' injuries were recorded and analyzed in most cases. The amputated specimens were dissected by the pathologist with a special view to correlation of the gross appearance of individual muscles with their microscopic changes. The various samples of muscle were embedded in paraffin and stained routinely with hematoxylin and eosin. A bacterial stain (Goodpasture's modification of the Gram stain) and connective tissue stains (Masson's trichrome and phosphotungstic acid hematoxylin stains) were done in selected cases. All muscle sections were viewed using polarized light. Chemical determinations of myoglobin and hemoglobin content were performed on one specimen.

Results

Muscle Changes During the First Two Days After Arterial Injury. Thirteen samples of muscle were taken from 11 soldiers at intervals of 6.5 to 27 hours after arterial injury. In one (Case 11), the specimen was removed from an amputated limb. In all others, it was obtained by biopsy. The salient clinical and pathologic features of these cases are presented in Table 1.

Surgical Appearance of Muscles. Four of the soldiers (*see* Cases 1, 6, 7, and 11) had muscle contractures resembling rigor mortis. The involved muscles were described as being hard, tight, and fixed. In four soldiers (*see* Cases 7, 9, 10, and 11), two of whom had contractures, the muscles were considered by the surgeon to be severely ischemic and probably irreversibly damaged. These muscles exhibited one or more of the following abnormalities: a soft or mushy consistency, a pale or bluish-gray color, a failure to bleed or delayed bleeding on incision, and an inability to contract on pinching. The muscles in the remaining five cases appeared normal at operation.

Pathologic Appearance of Muscles. Many of the specimens, irrespective of their appearance at operation, exhibited artefacts or changes attributable to nearby trauma rather than to ischemia. The more common artefacts were irregular transverse cracking of fibers, blurring of cross-striations, separation of fibers from their sarcolemmae and from one another, swelling with loss of structure of sarcoplasm, and nuclear shrinkage. The last three findings were considered of no significance only when they appeared near the edges of the specimens. Changes that were interpreted as being manifestations of nearby trauma were separation of fibers and swelling of muscle and endothelial nuclei (Fig. 1).

Table 1. Summary of Muscle Changes After Arterial Injury

Case	Type of Vascular Injury*	Appearance of Limb	Muscle Biopsied	Surgical Appearance of Muscle	Hours Before Biopsy	Significant Microscopic Findings	Results
1B	lac. right sup. fem. a.	cold, blue	ant. tib.	contracture	6½	rare congested small vessels; small foci of hemorrhage	muscle result good; renal failure; death—10 days
1C			gastroc.	contracture	6½	rare small foci of hemorrhage	
2G	sev. left sup. fem. a.	cold, pale	gastroc.	normal	7½	no abnormalities	muscle result good
3	perf. right pop. a.; sev. rt. pop. a.	cold, white	gastroc.	normal	9	focal prominence sarcolemmal & endothelial nuclei (near wound)	immediate result good; amp.: v. graft throm. due pressure bone frag.—5 days
4B	lac. right brachial a.	cool; good color	biceps	normal	9½	congested small vessels; occasional small hemorrhages	excellent
5	lac. left femoral a.	warm; normal color	?	normal	10	focal interfibrillar edema (near wound)	excellent
6C	lac. left pop. a.	cold, white	gastroc.	contracture: hard, spastic	10	no abnormalities	excellent

7	sev. left pop. a. and vein	swollen, cold and white	gastroc.	early contracture; non-bleeding; "nonviable"	11½	exaggeration of cross-striations; capillaries prominent	amputation: thrombosis of anastomosis—6 days
8D	lac. left pop. a.	cold; mot-tled with blue	ant. tib.	-----	12½	many fibers separated (near wound)	excellent
9	sev. right pop. a.	swollen, cold and blue	gastroc.	"nonviable"	15½	separation of fibers; venous & cap. engorgement (? thrombosis); exaggeration of cross-striations	died 2+ days after injury; leg remained in poor condition
10C	sev. left pop. a (also had sev. of right pop. a.)	left foot —early gangrene	gastroc.	ischemic	26	intense poly. infiltration, vein walls	died on operating table of pulmonary edema
10D			ant. tib.	"nonviable"	26	exaggeration of cross-striations	
11	throm. sup. femoral a.	swollen	ant. tib.	contracture; "nonviable"	27	separation of fibers; venous & cap. engorgement; exaggeration of cross-striations; focal arteritis	amp. 2 days after biopsy

*Abbreviations: a. artery
c. capillary
v. vein
amp. amputation
frag. fragments
lac. laceration
perf. perforation
sev. severance
thromb. thrombosis

Changes of questionable etiology, possibly related to ischemia in some of the specimens, were focal congestion of small blood vessels and perivascular petechiae.

Excluding the above changes, the five samples of muscle considered normal by the surgeon (*see* Cases 2, 3, 4, 5, and 8) were also unremarkable microscopically. Biopsies from the three muscles which were in contracture but which did not appear ischemic at operation (*see* Cases 1 and 6) were likewise essentially normal under the microscope (Fig. 2).

Of the five specimens of muscle regarded as severely ischemic by the surgeon, two (*see* Cases 7 and 10, Specimen D) showed a subtle but definite exaggeration of cross-striations as the only significant changes (Fig. 3). The striations, in addition to appearing coarser than usual,

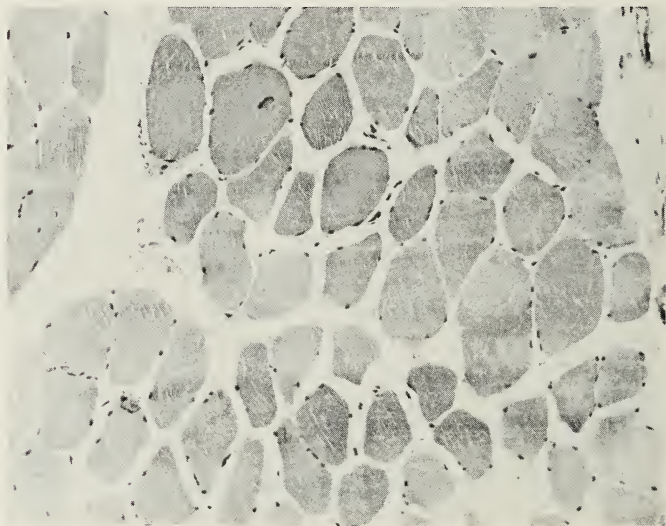


FIGURE 1. Case 3. Focal separation of fibers with prominence of muscle and endothelial nuclei. This is interpreted as being due to proximity to a wound.

were often smoothly curved instead of straight. One "severely ischemic" muscle (*see* Case 10, Specimen C) was remarkable only for the presence of intense polymorphonuclear leukocytic infiltration in the walls of its veins. Finally, two muscles placed in the severely damaged category by the surgeon (*see* Cases 9 and 11) showed marked changes histologically. These comprised, in addition to exaggeration and curving of cross-striations, separation and individualization of fibers and striking engorgement (? thrombosis) of small veins and capillaries with erythrocytes (Fig. 4). The specimen of Case 11 further exhibited focal necrosis of several medium-sized arteries. The necrotic lesions were characterized by fibrinoid degeneration, slight

polymorphonuclear infiltration, marked edema of the vessel walls, and a spread of round cells into the perivascular connective tissue. These changes resembled strongly those seen in periarteritis nodosa.

Loss of wavy arrangement of longitudinal fibrils described by Harman¹ as an early manifestation of experimental muscle ischemia, characterized all the "severely ischemic" muscles in the series. However, this change was also observed in several of the muscles which appeared otherwise normal at operation and under the microscope.

Muscle Changes During the First Two Days After Surgical Repair of Arterial Injury. Five specimens were obtained by biopsy from five soldiers at intervals of 24 to 56 hours after arterial injury (and 12 to 48 hours following surgical repair). The salient details of these cases are presented in Table 2.

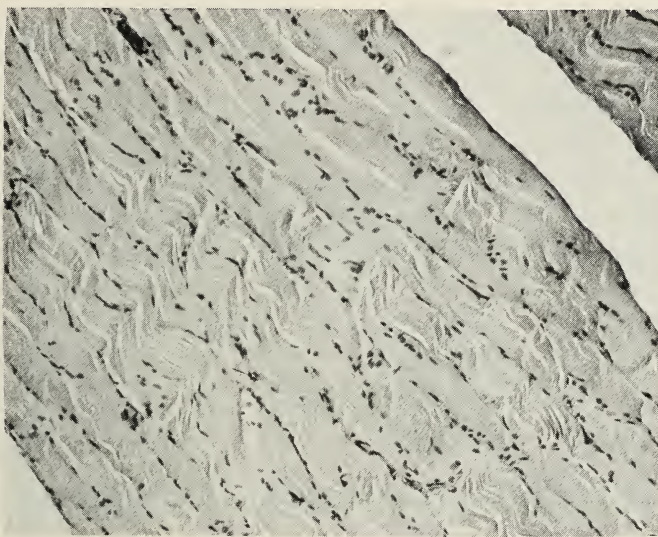


FIGURE 2. Case 4. Histologically normal muscle. Note close approximation of fibers and wavy character of fibrils.

Surgical Appearance of Muscles. In four of the five soldiers a striking clinical feature was the onset of muscle swelling some hours after vascular repair. When fasciotomies were performed to relieve the tension within the muscle compartments, the swollen muscles bulged through the incisions. In some instances, blood-tinged fluid exuded. In addition to swelling, all four swollen muscles exhibited changes suggestive of severe ischemia. In Case 16, the muscle was not swollen and appeared normal at operation.

Pathologic Appearance of Muscles. Microscopically, the biopsy sample of Case 16 was essentially normal. The specimens of the four swollen and "ischemic" muscles, on the other hand, exhibited striking

Table 2. Summary of Muscle Changes After Arterial Repair

Case	Type of Vascular Injury *	Type of Repair and Hours After Injury	Appearance of Limb at Biopsy	Muscle Biopsied	Surgical Appearance of Muscle	Hours—Injury to Biopsy	Microscopic Findings	Outcome
12	sev. left brachial a.; throm. of prof. a.	anas. & removal of throm.; lig. of br. veins—12 hours relief of spasm; lig. of prof. a.; repair, fem. v.—11 hours	warm, but swollen	flexor comp. part. muscle	contraction; swollen; bulging; bled slowly ischemic; swollen	24	slight separation, many fibers; exaggeration of cross-striations; focal waxy swelling	exc. of flexor compart. 16th day after biopsy
13A	spasm, left com. fem. a.; sev. prof. a. (also exp. to cold)	vein graft; lig. ax. veins—9½ hours	swollen; pulseless	gastroc.		33½	marked focal waxy & vacuolar degeneration; focal exaggeration of cross-striations	amp. on 3d day after biopsy
14	sec. left ax. a. & prof. a.		?	flexor comp. part. muscle	hard, knotted; edematous, "ischemic"; bled slowly	33½	advanced amorphous necrosis, disruption, and poly. infiltration	throm. of graft; result poor

15D	lac. right sup. fem. a.	anas. a— 12½ hours	warm	ant. tib.	ischemic; swollen	46	exaggeration of cross-stria- tions; many large & small hemorrhages; cap. promi- nent; congested small veins essentially normal	exc. of ant. tib. compart. 26th day after bi- opsy muscle result good
16	sev. right pop. a. (2); sev. right fem. a.	anas. (3); lig. fem. v.— 8 hours	warm; good col- or	gastroc.	negative	56		

* Abbreviations: amp. amputation
 anas. anastomosis
 exc. excision
 exp. exposure
 lac. laceration
 lig. ligation
 poly. polymorphonuclear
 sev. severance
 throm. thrombosis

changes. Because all four cases presented special features of interest, they merit separate brief discussion.

In Case 14, the biopsy specimen showed advanced necrosis characterized by disruption and loss of structure of fibers, and extensive polymorphonuclear leukocytic infiltration. These changes were far out of proportion to and different in kind from those ordinarily observed in ischemic muscle. They were also quite similar to those characterizing directly traumatized muscle. Therefore, this specimen was interpreted as having been taken from contused muscle, even though the surgeon was able to find no clinical evidence of this.

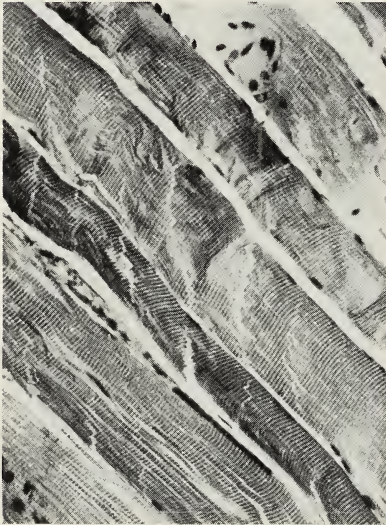


FIGURE 3. Case 7. Exaggeration of cross-striations. (For pictorial purposes, filter was used to emphasize the striations.)

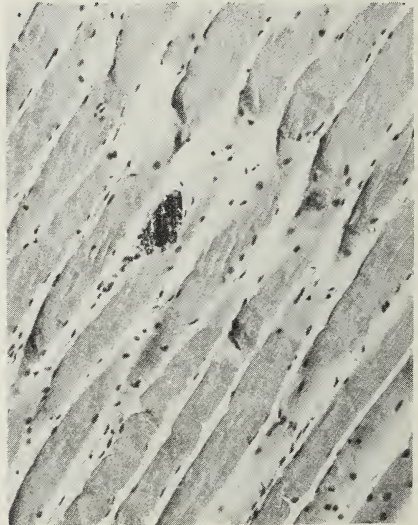


FIGURE 4. Case 11. Separation and individualization of fibers; engorgement (? thrombosis) of small vein.

In Case 15, the anterior tibial compartment gradually swelled after repair. A fasciotomy, performed 34 hours following operation, revealed swollen muscle which appeared severely ischemic. Microscopically the biopsy specimen showed changes consistent with early ischemia; exaggeration of cross-striations, prominent capillaries, congested small vessels, and numerous large and small hemorrhages.

In Case 13, pulsations were palpable in the foot following repair; later they disappeared as the calf muscles began to swell. At the time of fasciotomy, the gastrocnemius muscle was swollen and was considered probably nonviable. Microscopically most of the fibers were closely approximated. Over half of them showed degenerative changes. These included waxy swelling characterized by deep staining

of sarcoplasm, nuclear pyknosis, and focal fiber rupture (Zenker's degeneration or necrosis), vacuolar degeneration (Figs. 5 and 6), exaggeration of cross-striations, and weak staining of fibers with eosin. Many fibers were normal in appearance.

In Case 12, the muscles had been in contracture prior to operation. Although warmth was restored to the arm by vascular repair, the contracture increased thereafter, and the muscles of both flexor and extensor compartments began to swell. The small biopsy specimen showed slight separation of many fibers, exaggeration and slight curving of cross-striations, and small foci of waxy swelling of fibers.

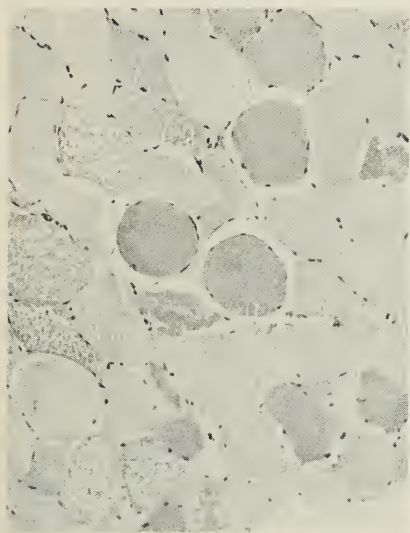


FIGURE 5. Case 13. Focal waxy and vacuolar degeneration of gastrocnemius muscle. Note that many of the fibers appear normal.

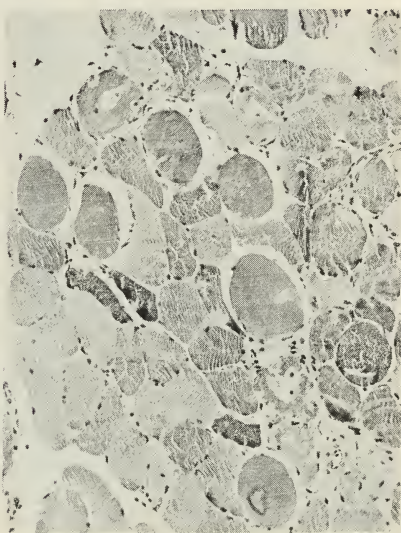


FIGURE 6. Case 13. Gastrocnemius muscle. Focal waxy and vacuolar degeneration. Most of the fibers appear normal.

Later Muscle Changes (Four to Twenty-six Days After Arterial Injury). The pathologic material obtained from 21 soldiers included five biopsy specimens, three specimens composed of fragments from muscle compartment excisions, six samples taken from amputated limbs, twelve entire amputated limbs, and one amputation stump.

Surgical Appearance of Muscles. Adequately detailed data unfortunately were not available on this aspect of most of the cases. Although the surgeon's observations must have been similar to the gross observations of the pathologist in some instances, it is obvious that such properties as color and consistency may often have differed at operation and at the dissecting table. Therefore, the gross pathologic findings described below cannot be regarded as coinciding in every respect with the findings of the surgeon.

Pathologic Appearance of Muscles. A wide range of pathologic changes characterized the later stages of muscle ischemia. Essentially four types of muscle were seen. Categorized in order of decreasing damage, these were: (1) more or less completely necrotic muscle with little or nothing in the way of inflammatory or reparative responses, (2) muscle showing patchy, and usually extensive necrosis, but exhibiting inflammatory and reparative responses as well, (3) severely damaged muscle showing small foci of complete necrosis, but notable for widespread survival of stroma and muscle regeneration,

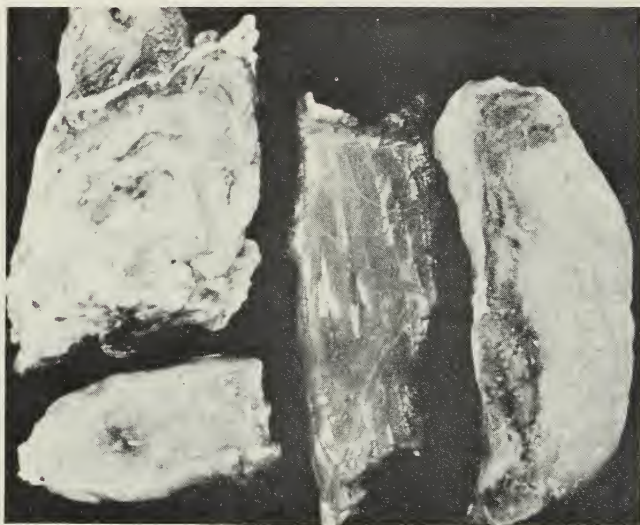


FIGURE 7. Case 26. Photograph of portions of muscles of the leg. The soleus muscle (upper left) shows a characteristic pattern of patchy pale brown zones alternating with irregular broad, yellowish-white bands. The flexor hallucis longus muscle (lower left) and extensor hallucis longus muscle (center) are of a normal or at most a slightly pale color. The lateral head of the gastrocnemius (right) is for the greater part the color of fat. At the left is a rim of pale red surrounding blotchy opaque brownish-yellow tissue. See corresponding photomicrographs (Figs. 5, 6, 9, 10, 14, 15).

and (4) essentially normal or minimally damaged muscle. Although borderline forms existed between these four categories and although one portion of a given muscle might fit into one category and another portion into a second, by and large an entire muscle or a large part of it was uniform in its pathologic appearance.

1. Completely necrotic muscles were most often exemplified by the long slender muscles of the leg. On dissection of the amputated limb, these muscles appeared to be of normal color or somewhat pale (Fig. 7). Their consistency was normal or slightly flabby. They were at times swollen and bulged slightly when their fasciae were

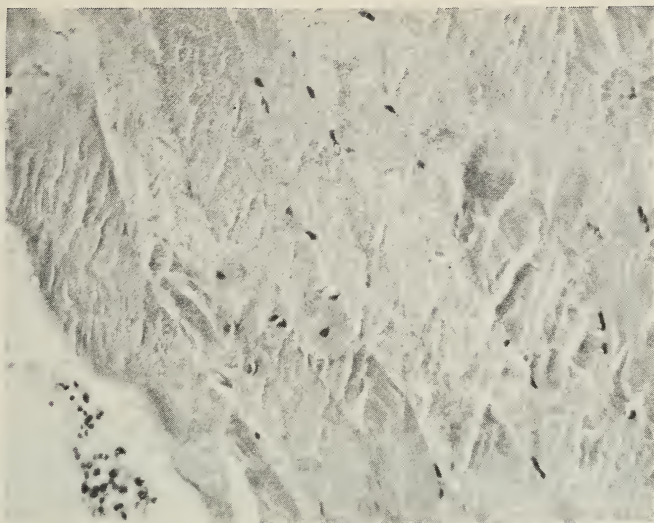


FIGURE 8. Case 22. Gastrocnemius muscle. Granular disintegration of muscle fibers.

incised. Microscopically there was extensive necrosis involving both fibers and interstitial tissue. The fibers were closely approximated in some instances (Figs. 8 and 9), widely separated in others (Fig. 10). Although some fibers had structureless sarcoplasm and appeared slightly swollen, the predominant change was one of discoid necrosis.



FIGURE 9. Case 26. Flexor hallucis longus muscle. Fragmentation and granular disintegration of fibers.

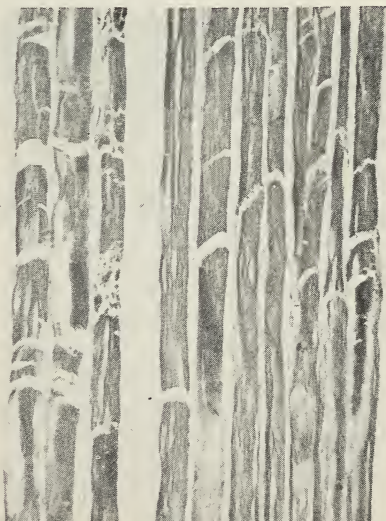


FIGURE 10. Case 26. Extensor hallucis longus muscle. Discoid degeneration with curving of discs and cracking between them. Nuclei have disappeared.

Many of the discs were curved and there was periodic transverse cracking of the fibers between them (Fig. 10). The sarcoplasm often stained feebly with eosin. The nuclei showed increasing degrees of shrinkage and disappeared in time. In the areas of most advanced damage, the fibers were split longitudinally and transversely or were fused into coarsely granular amorphous masses (*see* Fig. 8). The interstitial tissue showed necrosis as evidenced by its weak staining properties and shrinkage of nuclei. The small vessels were collapsed and their contents were no longer recognizable as blood. There was no congestion; at most, an exceedingly thin band of polymorphonuclear leukocytes was the sole evidence of an inflammatory response. A frequent finding in this type of muscle, if it lay in proximity to a

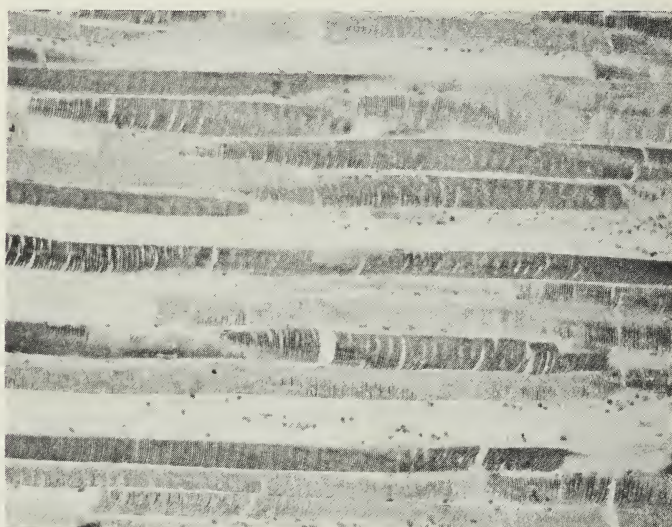


FIGURE 11. Case 23. Discoid necrosis of muscle of flexor compartment of arm. Many of the fibers fail to show the normal dark blue coloration with phosphotungstic and hematoxylin stain. The fibers are individualized and show curving of and cracking between the discs.

wound or infected incision, was an invasion by bacteria sometimes in massive numbers. Some of these bacteria were morphologically recognizable as *Clostridia* species. They appeared in the perimysium, endomysium, and within the sarcolemmae. In our small series, gas, edema, or other recognizable lesions attributable to the presence of *Clostridia* were not seen.

2. Patchy and usually extensive necrosis, inflammation, and repair were observed most often in the soleus muscle (*see* Fig. 7); other muscles occasionally showed this type of change. Grossly these muscles were brownish-yellow or had such a light yellow color that they were easily mistaken for fat. A common appearance was a geographic pattern of pale brown patches of necrosis separated by yellow

or white bands of inflammatory exudate (*see* Fig. 7). Gross areas of hemorrhage were often visible. Although the consistency was generally about normal, it was sometimes mushy or liquid. Microscopically, there was extensive patchy death of both fibers and interstitial tissue. The fibers sometimes showed a more or less pure picture of discoid necrosis (Fig. 11); more often, however, there was a considerable admixture with swollen fibers having blurred or absent striations. It was also not uncommon to find dead fibers which had retained exceedingly delicate cross-striations. The fibers were generally separated from one another, sometimes by empty spaces (Fig. 11), at other times, by edema fluid and disintegrating polymorphonuclear leukocytic exudate. If the muscle was liquefied, fragments of

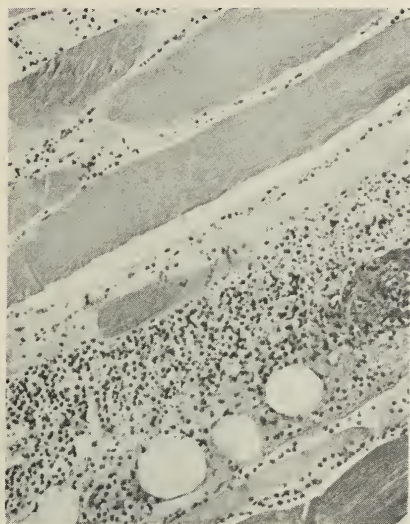


FIGURE 12. Case 13. Soleus muscle. Swollen homogenous, anuclear, necrotic fibers are separated by dense polymorphonuclear leukocytic exudate. Note thrombosed vein.

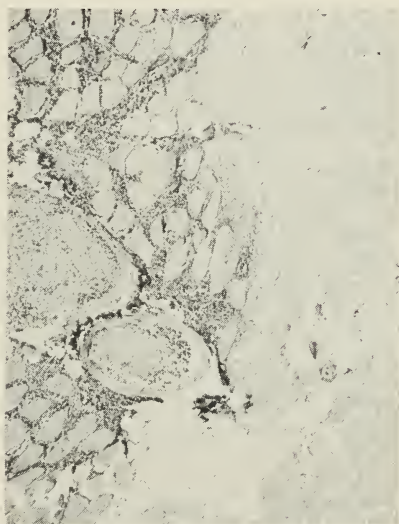


FIGURE 13. Case 26. Soleus muscle. Note dead muscle on the right. On the left there is extensive polymorphonuclear infiltration of dead muscle. A large thrombosed vein is visible at the center.

disintegrating fibers penetrated by polymorphonuclear leukocytes might be seen lying in seas of exudate or hemorrhage. The interstitial tissue in this category of muscle stained weakly and exhibited pyknosis of its nuclei. The small vessels were collapsed and necrotic or were distended by closely packed erythrocytes. The larger vessels in and near the zones of necrosis commonly showed inflammation or necrosis of their walls. Often the veins, and less frequently the arteries, were distended by thrombi (Figs. 12 and 13) which eventually underwent organization.

In the muscle peripheral to the areas which had undergone complete necrosis, a variety of changes was seen. Early, bands of disinteg-

ing polymorphonuclear leukocytes characterized this zone (Figs. 12, 13, and 14). Later, histiocytic invasion of sarcolemmic tubes with digestion of degenerating sarcoplasm, and an addition of chronic inflammatory cells were prominent features. Finally, there appeared a striking proliferation of capillaries and of fibroblasts laying down collagen (Fig. 15), as well as an intense but limited muscle cell regeneration. We have not observed more than minimal penetration of fibroblasts, capillaries, and regenerating muscle nuclei into completely necrotic muscle, a process which has been described as extensive in rabbits with experimental ischemic muscle necrosis.²

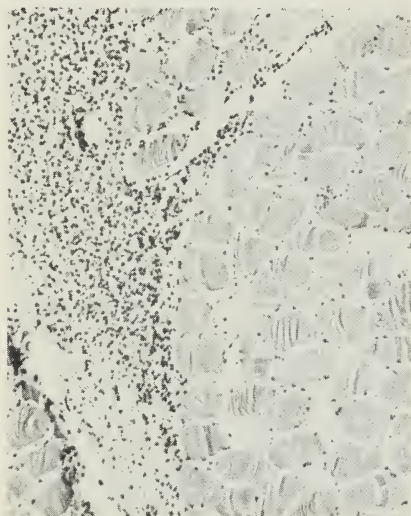


FIGURE 14. Case 26. Lateral head of gastrocnemius muscle corresponding to opaque brownish-yellow area. Note discoid necrosis and separation of fibers and broad zone of polymorphonuclear leukocytic infiltration in perimysium.

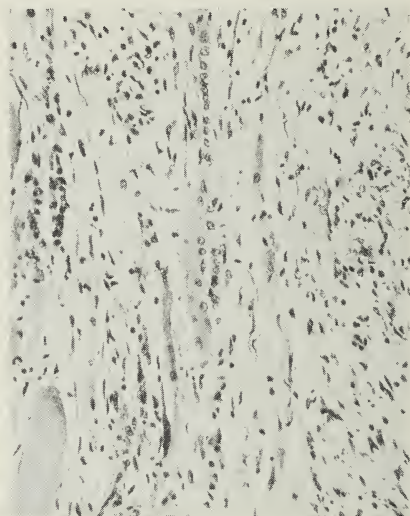


FIGURE 15. Case 31. Note necrotic, swollen muscle fibers at left, and dense fibrous tissue at right. Near the center are a few regenerating muscle fibers.

In one case (Case 31) the myoglobin content of a pale yellow muscle belonging in this category was analyzed; and it was found to be half its normal value.

3. Severely damaged but live muscle with widespread regenerative activity was exemplified in two cases (*see* Cases 23 and 26); in the first, by the triceps (8 days after wounding) and, in the second, by the gastrocnemius (10 days after wounding). The soleus muscle of Case 29 and the upper portion of the gastrocnemius muscle of Case 30 appeared to belong in Category 3 from a microscopic viewpoint. However, since no detailed gross descriptions were given, and since the samples taken for microscopic examination possibly may not have been representative of any large portions of the muscles, these were

not definitely placed in this category. The triceps (Case 23) was cream-colored with focal patches of hemorrhage. The gastrocnemius (Case 26), in large part, presented a color somewhat paler than that of fat (*see* Fig. 7). Both muscles were of more or less normal consistency. Microscopically these muscles showed severe degenerative changes within the fibers, but a survival of many muscle nuclei and of interstitial tissue. Since complete necrosis occurred, at most, in small foci, there was little stimulation to fibroblastic proliferation, and eventual extensive muscle regeneration and reconstitution seemed possible. The degenerating sarcoplasm was discoid, structureless, or

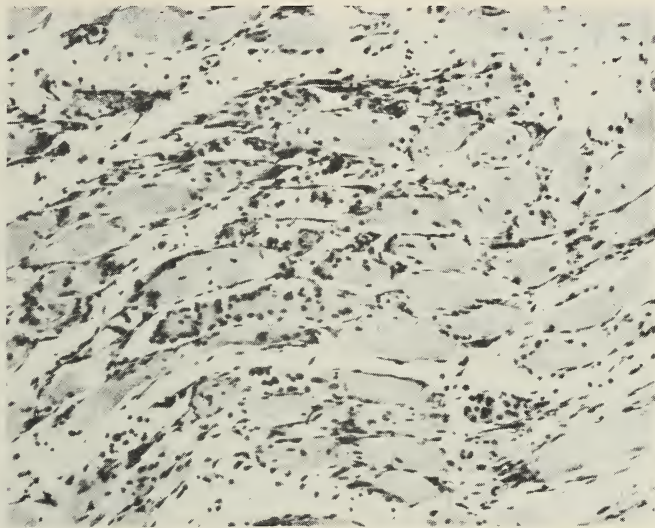


FIGURE 16. Case 23. Triceps muscle. Fibers show disintegrating sarcoplasm. Some sarcolemmic tubes contain large numbers of histiocytes. Most of the fibers are surrounded by necklaces of elongated cells.

fragmented. The fibers were closely approximated or were separated from one another by an edematous endomysium containing scattered histiocytes and round cells. The perimysium was edematous and contained inflammatory cells in small numbers. Within the sarcolemmic tubes were focal collections of histiocytes in the process of digesting the degenerating sarcoplasm (Fig. 16). Along the edges of the fibers, nuclei were periodically missing. Elsewhere, however, were rows of elongated cells with scant cytoplasm (Figs. 16, 17, and 18). Some of these were recognizable as regenerating muscle cells with basophilic cytoplasm; others as endothelial cells of elongated tubular capillaries (Fig. 18).

What appeared to be later phases of the same process microscopically were observed in Cases 29 and 30 (18 and 12 days after wounding, respectively). These muscles showed large numbers of thin regenerating fibers arrayed in an orderly fashion in a very edematous

viable connective tissue stroma (Figs. 19 and 20). The degenerated fibers were no longer recognizable and the only trace of previous damage was the presence of variable numbers of lymphocytes, plasma cells, and histiocytes in the stroma. The new fibers had longitudinal fibrils; in a few of them cross-striations could be identified.

4. Essentially normal muscle. Of the muscles examined microscopically, the gastrocnemius or large portions of it most commonly fell into this category. This type of muscle sometimes exhibited focal fiber damage in the form of waxy swelling or vacuolar degen-

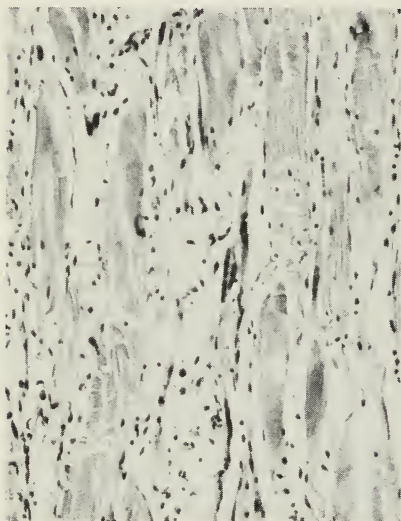


FIGURE 17. Case 26. Lateral head of gastrocnemius, corresponding to pale yellow area grossly. Note elongated bands of regenerating cells among degenerating fibers.

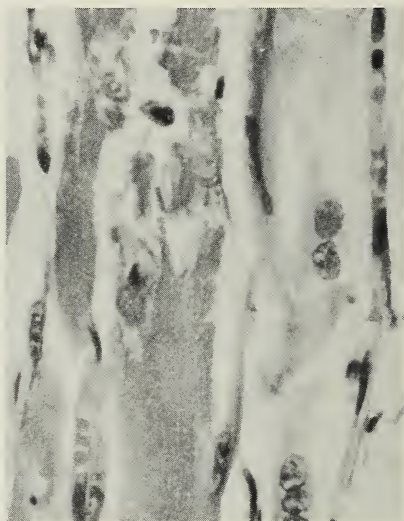


FIGURE 18. Case 26. Lateral head of gastrocnemius muscle. Note disintegrating sarcoplasm, elongated capillary tubes, and regenerating multinucleated muscle cells at top and midcenter.

eration. Small areas of inflammation, regeneration, and repair were also seen.

A striking finding in the amputated legs was the relatively better condition of the gastrocnemius than the soleus muscle after the major artery of supply to the extremity had been damaged. Thus, of 10 cases in which damage occurred to either the femoral or popliteal artery and in which sections of both gastrocnemius and soleus muscles were available for study, the former showed a lesser degree of ischemic change in nine and a slightly greater degree in only one. In some instances, the gastrocnemius appeared relatively normal when the soleus was either necrotic or regenerating; in others, the former was regenerating when the latter was necrotic; while in

still others the former, though extensively damaged, exhibited a somewhat less complete form of necrosis than the latter.

Because of the nature of the material, it was not possible to relate the pathologic category into which individual muscles fell to lesions in specific arteries or veins or to explain why certain muscles fared better than others. This aspect of the pathology of muscle ischemia will be discussed at greater length below.

The use of special connective tissue staining and of polarizing microscopy afforded additional data on the microscopic characteristics

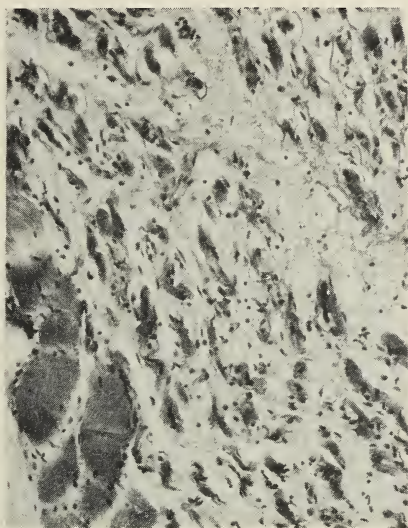


FIGURE 19. Case 29. Soleus muscle. At the left are some remaining normal fibers. To their right are numerous small regenerating fibers arrayed in an orderly fashion in edematous connective tissue. Small numbers of chronic inflammatory cells lie among the small fibers.

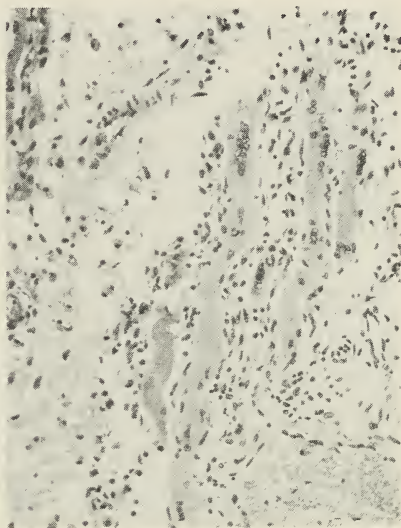


FIGURE 20. Case 30. Gastrocnemius muscle. Elongated regenerating fibers with multiple clusters of nuclei are separated by histiocytes, chronic inflammatory cells, and edema fluid.

of ischemic muscle. With phosphotungstic acid hematoxylin and Masson's trichrome stains (using aniline blue for the latter), degenerating and necrotic muscle fibers often failed to show normal staining properties. These damaged cells were weakly colored or exhibited atypical colors, such as blue with Masson's or buff with PTAH stain (*see* Fig. 7). Waxy fibers, swollen necrotic fibers, discoid fibers, and the degenerating sarcoplasm of the muscles of Category 3 commonly showed weak or atypical staining. Although Mallory's two cases of crush syndrome suggested an association of these abnormal staining properties with myoglobin loss,³ we found them in normally

pigmented muscles as well as in depigmented ones. Regenerating muscle cells have stained normally with the connective tissue stains in our limited experience.

Using polarized light, it was found that muscle fibers commonly retained their birefringence in advanced stages of necrosis. Indeed, swollen fibers and fibers showing discoid necrosis were often brightly refractile even after their nuclei had completely disappeared. Refractility was lost in some fibers showing advanced discoid or structureless necrosis and in fibers exhibiting granular disintegration. It reappeared in the early regenerating fibers of Cases 29 and 30.

Discussion

Since most of the basic knowledge of muscle ischemia has come as a result of animal experimentation, a comparison of experimental findings with observations on human cases is in order. Such a comparison may suggest which experimental conclusions can be applied justifiably to humans and what type of further experimentation may be expected to yield clinically valuable information.

The first manifestation of muscle ischemia following arterial injury in humans is contracture. Early ischemic contracture is not to be confused with Volkmann's ischemic contracture, a permanent contracture associated with muscle infarction. It is possible, however, that early ischemic contracture represents an early, reversible stage in the development of Volkmann's contracture. In our series, no instances of persistent contracture were encountered. Early ischemic contracture may have its onset as early as 6 or 7 hours after injury. The muscles of the flexor compartment of the forearm and of the anterior tibial compartment of the leg are most prone to be affected. The contracture is reversible, at least in its initial states. It is not an invariable manifestation of muscle ischemia. In the present small series, the nature of the vascular damage did not seem to differ in those soldiers developing contracture and in those free from it. Likewise, the application of tourniquets did not appear to be a factor. No structural counterpart for early ischemic contracture was identified in the human specimens. In the earlier cases in which the muscles involved appeared otherwise normal at operation, microscopic examination of the samples was essentially unremarkable. In later cases in which the muscles exhibited other evidences of ischemia at surgery, the biopsy specimens showed changes similar to those seen in ischemia without accompanying contracture. Early ischemic contracture has been produced experimentally in muscles in which the arterial blood supply has been more or less completely interrupted;^{4 5} however, its nature has not been investigated. Harman, who has furnished de-

tailed data on the microscopic features of early muscle ischemia in animals,¹ has not mentioned contracture as an ischemic phenomenon.

The earliest structural manifestations of muscle ischemia observed in several humans were similar to those described by Harman in experimental animals (*i. e.*, exaggeration of cross-striations and separation and individualization of fibers).¹ Loss of wavy arrangement of longitudinal fibrils, however, did not prove to be a reliable sign of early ischemia in humans. Although this change characterized all the "severely ischemic" specimens, it was observed in several samples of otherwise normal muscle as well. The vascular engorgement (? thrombosis) observed in two of the human "severely ischemic" samples may have been the structural counterpart of the physiologic damage to small vessels demonstrated by Harman⁶ in experimental muscle ischemia. The usual vascular changes seen in Cases 10 and 11, *i. e.*, the leukocytic infiltration of the vein walls, and the necrotic arterial lesions resembling those of periarteritis, were unique in the series and, to our knowledge, have not been described in experimental ischemia. Interpretation of their relationship to ischemia must remain conjectural at the present time.

The correlation between surgeon's judgment at operation and the pathologist's interpretation based on microscopic examination, as to muscle viability in early ischemia, was unsatisfactory in the present series. In two specimens believed by the surgeon to be "nonviable" (*see* Cases 9 and 11) the histologic changes were so severe that a pathologist might justifiably question viability. In three other specimens considered severely ischemic at operation (*see* Cases 7 and 10), the microscopic abnormalities were, in contrast, only slight. In these two cases it is possible that severe or irreversible changes had taken place, but that the structural manifestations were still too subtle to be recognized. However, if Harman's morphologic criteria for muscle viability in experimental ischemia¹ can be transferred to human pathology, the muscles with slight changes were definitely viable and capable of recovery despite their striking abnormalities at operation. Unfortunately in the two cases in which the surgeon and pathologist disagreed (*see* Cases 7 and 10), the final outcome did not permit conclusions as to whether the muscles were actually viable or were dead at the time of biopsy. Although Harman has established microscopic criteria and has demonstrated the invalidity of contractibility as a criterion of muscle viability in animals, the relative values of other surgical criteria (*e. g.*, color, consistency, and ability to bleed) have not been investigated experimentally to our knowledge.

Swelling of ischemic muscles occurs in humans with arterial injury as well as in experimental animals. In humans it may take place in instances of unrelieved ischemia; however, it is more frequent and

more severe following surgical restoration of circulation. In experiments performed on animals, ischemic swelling may appear as a result of less than complete interruption of arterial blood supply⁷ or may follow release of arterial obstruction.^{5,7,8} Pathologic examination of human biopsy specimens of swollen muscle has failed to reveal the pathogenesis of the swelling. It seems possible that five factors, alone or in combination, may contribute to ischemic swelling: (1) lymphatic stasis, (2) vascular congestion, (3) enlargement of individual fibers, (4) interfibrillar edema, and (5) edema of the perimysium (connective tissue between bundles of fibers). Our biopsy specimens did not permit evaluation of the first factor. Vascular congestion and focal hemorrhages were observed only in Case 15. Enlargement of individual fibers was prominent in Case 13 and to a much lesser extent in Case 12. Interfibrillar edema, as evidenced by separation of fibers, was present to a slight degree in Case 12. Neither vascular congestion nor enlargement of individual fibers was observed in Case 15. Studies in experimental animals have shed little light on the nature of ischemic swelling, although edema and hemorrhages have been described in the involved muscles.^{7,8} It is possible that larger biopsy specimens or micrometric measurement of muscle fiber size may provide an eventual answer to the problem.

The presence of a Zenker's type of muscle degeneration, not seen in early unrelieved ischemia but seen in two specimens of swollen muscle hours after restoration of circulation (*see* Cases 12 and 13), is most interesting in view of the fact that Harman⁹ found this change under similar circumstances in his experimental animals. In the small biopsy specimen of Case 12, this waxy type of degeneration was present only in a few foci. In the specimen of Case 13, the change was widespread. Here, however, the soldier had been exposed to cold, and since fiber degeneration similar to Zenker's has been reported in experimental hypothermia⁹ as well as in relieved ischemia, cold injury cannot be entirely ruled out as the etiologic factor. If the unusual changes of Cases 12 and 13 are due to restoration of circulation, Harman's observations on the increase in muscle fiber damage wrought by a return of circulating blood may have a direct application to vascular repair in humans. The occurrence of muscle swelling and of new microscopic types of muscle damage following resumption of blood flow in an ischemic muscle also calls attention to the two experiments of Brooks⁷ which suggested that a gradual or incomplete return of circulation might result in less damage to an ischemic muscle than an abrupt and complete one.

Wide varieties of pathologic changes, ranging from minimal damage to massive necrosis, characterize the later stages of muscle ischemia, both clinical and experimental. For the most part, the findings in man and animal have been quite similar. Two aspects of the

subject in which clinical and experimental observations are somewhat at variance, namely regeneration and depigmentation, deserve special attention.

Clark has described extensive muscle regeneration and reconstitution in small experimental animals even when the degree of arterial ischemia has been so great that necrosis of the interstitial tissue has taken place.² The same author, however, is quoted as saying that a similar degree of regeneration would appear unlikely in man because of the bulk of human muscles.¹⁰ Although we have seen only abortive attempts at regeneration when severe necrosis involving the interstitial tissue has occurred, several of our human specimens in which the muscle fibers had undergone widespread degenerative changes, but in which the stroma had survived, showed such striking regeneration that a considerable amount of reconstitution seemed likely had the limb remained intact. More extensive experience with human cases are needed to confirm and expand these observations.

Although Montagnani and Simeone have demonstrated liberation of myoglobin from muscles upon release of ischemia,¹¹ visible depigmentation as a phenomenon of experimental muscle ischemia has not, to our knowledge, been described. Our studies have shown that in humans severe and widespread loss of pigment may occur not only in muscles which show large areas of necrosis, but also in those exhibiting regenerative changes. Thus the appearance of fish-flesh, cream, or pale yellow color in a muscle does not necessarily indicate that it is irreversibly degenerated. In our limited experience, muscle which is more or less completely deprived of its blood supply does not undergo depigmentation, perhaps for the reason that no circulatory system exists in the muscle to furnish enzymes necessary for the release of the myoglobin and to provide for transportation of this pigment from the muscle.

Whereas it is difficult to determine in experimental animals what pathogenic factors are responsible for producing the various forms and degrees of ischemic muscle degeneration, this is an even greater problem when dealing with human cases. Here the variables are multiplied many fold and investigative methods are necessarily limited in extent. Thus, in our series, often only a biopsy specimen was available for study. This may not always have been representative of an entire muscle, nor did it afford information about the state of the blood vessels supplying the muscle. In cases where an entire amputated limb was forwarded for examination, usually the obstructed major vessel remained in the patient above the amputation site. Spasm of arteries or veins and the degree of obliteration of collateral vessels were most difficult, if not impossible, to evaluate. Many of the soldiers had had tourniquets applied; although in most cases in-

formation was given as to the duration and periods of release, it was not possible to determine how effectively the arterial flow had been obstructed. Exposure to excessive cold or heat, the state of fatigue of the muscle at the time of onset of ischemia, and individual variations in vascular supply were some of the other factors which did not lend themselves to accurate determination.

Considering all these unknown and variable factors, plus the relatively small size of our series, we are unable to state the precise roles of arterial, venous, and capillary obstruction in producing the muscle changes observed. In addition to arterial damage, large veins were directly injured or were ligated in many of our cases. Moreover, major and intramuscular veins and capillaries were often extensively thrombosed in the pathologic specimens. In some instances, the thrombi formed in infarcted vessels damaged by ischemia which damaged the muscles in which they lay. It has been suggested that extensive venous thrombosis may produce muscle death in patients whose arterial flow has been adequately re-established. However, in one of our patients, the involved muscle groups with patent arterial supply and thrombosed veins did not show the pathologic picture encountered in ischemia due to experimental venous occlusion.

The early muscle changes observed in the present series are natural forerunners of the later changes, infarcts enclosed by zones of fibrous tissue, reported by previous authors: Bowden and Gutmann (Group 1), Griffiths.^{12 13} The extensive regeneration seen in our Category 3 muscles, to our knowledge, has not been described in humans.

There is a remarkable similarity between the pathologic changes in muscle ischemia secondary to arterial trauma and in that characterizing the crush syndrome. In the latter condition, muscle swelling, discoid necrosis, atypical staining of fibers with connective tissue stains, and regeneration have all been described. Moreover, gross depigmentation of muscle and even renal failure may be seen in cases of arterial injury without crushing.¹⁴ Pathologically, the striking similarity of the two conditions support other evidence that the muscle changes of the crush syndrome and due to arterial spasm.^{14 15}

Precipitation of granular material suggesting calcium, such as is seen in the degenerating muscle fibers involved in the crush syndrome, is unusual except in relation to wounds with ischemia following arterial trauma. The disordered metabolism of renal insufficiency may be the factor determining the presence or absence of this change.

A final observation that merits brief discussion is the usually better outcome of the gastrocnemius than the soleus muscle in injuries of the femoral or popliteal arteries. The reason for this finding is not apparent from this study, or from the literature. Blomfield who has investigated the blood supply of the leg muscles by injection technics

at autopsy has stated that the gastrocnemius is served by a single artery; the soleus, on the other hand, has at least five arteries of supply.¹⁶ The author further stated that in local wounds of the calf, the gastrocnemius is more apt to undergo necrosis and secondary clostridial infection because of its less rich blood supply. The disparity between the outcome of the two muscles following local vascular damage and following injury to the major artery of the extremity deserves investigation. Possibly the artery supplying the gastrocnemius communicates by collateral channels with vessels arising above the level of obstruction of the femoral or popliteal artery. Again, the comparative tightness of the sheaths or fascial envelopes of the two muscles or a difference in their metabolic requirements may play decisive roles in their responses to ischemia. A practical corollary of the observation regarding the gastrocnemius and soleus is that the condition of the former muscle cannot be used as a guide to that of the other muscles of the leg.

Summary and Conclusions

1. The early pathologic changes in skeletal muscle following damage to major limb arteries were studied in 31 soldiers, most of whom had sustained injuries on the Korean battlefields. The material consisted of biopsy specimens, surgically excised necrotic muscles, and amputated limbs. The specimens were obtained from 6.5 hours to 26 days after injury.

2. The pathologic lesions observed paralleled those described in experimental muscle ischemia and appeared to be the natural forerunners of later changes reported in humans.

3. The earliest ischemic change recognized under the microscope was an exaggeration of cross-striations. Later separation of fibers and capillary and venular engorgement became evident.

4. Swelling of ischemic muscles following restoration of circulation occurred in several cases. The nature and pathogenesis of this swelling was not evident from study of human or experimental data.

5. After the acute phase of ischemia had passed, the muscles studied fell into one of four pathologic categories: (1) more or less complete necrosis, (2) patchy, but extensive necrosis with inflammation and repair, (3) severe damage of fibers, but survival of stroma and widespread muscle regeneration, and (4) normal structure.

6. A pale yellow or cream color probably attributable to loss of myoglobin was an outstanding feature of several muscles in Category 3 as well as in Category 2. Therefore, depigmentation is not a reliable criteria of irreversible damage in ischemic muscle.

7. The pathologic changes of muscle ischemia following arterial injury resembled closely those reported in the crush syndrome, adding

to the evidence that the muscle lesions of the latter condition are due to arterial spasm.

8. Because of the many imponderable variables in human cases of arterial obstruction, it was not possible to assess the exact role of arterial, venous, and capillary obstruction in producing the muscle changes observed.

9. Surgical criteria for irreversible damage in ischemic skeletal muscle have not been established on a sound basis and deserve further experimental investigation.

10. The gastrocnemius muscle fared better than the soleus in the great majority of cases of obstruction of a major leg artery. This result was the opposite of that reported in local wounds involving the immediate arteries of supply of the muscles. The vascular anatomic background for this was not clear. Because of the difference in outcome of the two muscles it is apparent that the condition of the gastrocnemius cannot be used as a guide to that of the other muscles of the leg.

Illustrative Cases

Case 18. This 21-year-old soldier sustained a mortar-fragment wound which resulted in thrombosis of his right brachial artery. Four days after injury, the arm was amputated above the elbow because of progressive wet gangrene. Pathologic examination revealed moderate swelling of the forearm and hand, dark green discoloration of the skin and subcutaneous tissue of the forearm, and cyanosis of the finger tips. The muscles of the forearm were slightly pale and flabby, but not discolored or swollen. The brachial artery was thrombosed over a length of 8 cm., beginning at the amputation level; the radial and ulnar arteries were thrombosed for 2 to 3 cm. near their origins. The brachial vein and its tributaries, and several large superficial veins in the region of the elbow, were also thrombosed. There were two sizable avulsive wounds of the upper arm and two small superficial wounds of the forearm and antecubital space. Microscopically, multiple sections of superficial and deep muscles revealed striking ischemic changes. The fibers were universally individualized and separated, moderately so in some muscles, only slightly so in others. Some of the fibers showed exaggeration of cross-striations; in others the sarcoplasm was structureless, and the striations were blurred or absent; a few fibers were swollen. The muscle nuclei were not noticeably altered in some muscles, but were shrunken in others. The interstitial collagenous tissue and its nerves and vessels showed varying degrees of necrosis. They stained weakly with eosin and often their nuclei exhibited marked pyknosis. The small vessels were not congested but, for the most part, were collapsed; their lumens were empty or contained pale staining unidentifiable material upon which

basophilic debris was sometimes deposited. Here and there small collections of bacilli were found in the endomysium, perimysium, and beneath the sarcolemmae.

Comment. This type of necrosis, which is accompanied by little swelling of individual fibers and is unassociated with small vessel congestion or inflammatory cell reaction, is a reflection of a more or less complete interruption of arterial blood supply. Separation of fibers, which requires little if any intact circulation for its development in experimental animals,¹ was striking here; nevertheless, as in Case 11, it did not result in gross swelling of the muscles.

Case 13. The salient features of the early history have been presented in Table 2. Following fasciotomy (performed at the time of the original biopsy) the pulses failed to return and the foot remained cold. By the fourth day after wounding, the foot showed early gangrene. The leg was amputated below the knee. At operation it was noted that there was a good blood flow through the posterior tibial artery and its muscular branches, although the muscles appeared nonviable. Samples of both gastrocnemius and soleus muscles were taken from the amputated leg by the surgeon. The formalin-fixed gastrocnemius specimen taken at the fasciotomy site was pale tan with a zone of dark reddish-brown at one margin. Microscopically, one surface of the biopsy showed fascia, another showed granulation tissue containing an abundance of polymorphonuclear leukocytes. The muscle fibers close to the line of surgical incision were widely separated by the edematous granulation tissue. They showed various changes: loss of structure of sarcoplasm, vacuolar degeneration, swelling, rupture, and histiocytic digestion. The muscle at a distance from the granulating surface was essentially normal except for edema and focal hemorrhage of the perimysium, focal edema of the endomysium, swelling of muscle and endothelial nuclei, and spotty Zenker's and vacuolar degeneration (*see* Fig. 6).

The over-all picture was one of focally damaged muscle showing changes chiefly attributable to proximity to an operative incision. The formalin-fixed soleus specimen was dark brown, mottled with pale yellowish-gray. Microscopically, there was vast necrosis involving not only the muscle fibers, but their supporting tissue as well. The fibers were minimally to markedly separated. Many showed discoid degeneration, others loss of structure and swelling of their sarcoplasm with varying degrees of obscuration of cross-striations (*see* Fig. 12). The nuclei were absent or severely pyknotic. There was an extensive irregular outpouring of polymorphonuclear leukocytes and edema fluid. A thin zone just beneath the fascia showed a granulation tissue type of response. The larger vessels were often necrotic; many veins and fewer arteries contained thrombi (*see* Fig. 12). The

smaller vessels were frequently distended; they appeared dead and showed little affinity for stains.

Comment. There are several points of interest here: (1) the gastrocnemius which was thought to be nonviable at operation was essentially normal, except for focal fiber damage which might have been related, at least in part, to proximity to the fasciotomy incision. The amputation specimen exhibited less damage than the fasciotomy specimen which was taken three days before and was also suspected of being nonviable. (2) The soleus showed more extensive damage than the gastrocnemius.

Case 26. This 22-year-old soldier sustained multiple shell-fragment wounds, one of which severed his right superficial femoral artery and vein. On admission to a surgical hospital, the foot was cool and paralyzed and the thigh was swollen and tense. The artery was grafted, the vein ligated, and a calf fasciotomy performed 7.5 hours after injury. Postoperatively there was an adequate distal pulse. The day after the repair, the foot was cool and cyanotic. The next day no pulsation was felt and there was some edema of the foot and calf. Four days after wounding, demarcation was beginning at the mid-foot level, and the calf was tight and edematous. The next day an anteromedial fasciotomy was performed.

Eight days after wounding the leg was amputated above the knee. Pathologically there was considerable swelling of the leg; small areas of patchy bluish-pink discoloration were visible on the skin of the dorsum of the foot. Two long gaping fasciotomy incisions were present, as well as a smaller débrided wound. There was extensive thrombosis of the anterior tibial, posterior tibial, and peroneal veins. The corresponding arteries were free from thrombi. The muscles showed most interesting color changes. The lateral head of the gastrocnemius was pale yellow (not the intense yellow of fat, but a more washed-out color); in the peripheral portion of the muscle was a rim of red or pale red tissue alternating with blotchy opaque brownish-yellow areas (*see* Fig. 7).

The pale-yellow zone corresponded microscopically to a digestive and regenerative process more advanced than that seen in Case 23 (*see* Fig. 17). Here, the fibers were universally separated from one another; most of them showed discoid degeneration and a few were swollen and structureless. The sarcoplasm was being digested focally by histiocytes; and there was early regeneration of muscle cells with formation of many thin basophilic fibers (*see* Fig. 18). The endomysium and perimysium showed marked edema and infiltration with inflammatory cells, particularly histiocytes and round cells. The endomysial capillary network was prominent, often appearing in the form of longitudinal tubes extending between the muscle fibers (*see* Fig. 18). Corresponding to the blotchy opaque brownish-yellow areas

with red rims were microscopic zones of massive necrosis of discoid type with rims of granulation tissue. The necrosis involved both fibers and interstitial tissue and was accompanied by an outpouring of edema fluid and degenerating polymorphonuclear leukocytes (*see* Fig. 14). The medial head of the gastrocnemius in part resembled the lateral head, being light yellow and in an early regenerative state. Elsewhere, it was pale brown and exhibited extensive discoid necrosis with exudation of polymorphonuclear leukocytes and edema fluid. The soleus, on section, showed patchy opaque pale-brown zones alternating with irregular yellowish-white bands (*see* Fig. 7).

Microscopically, there was massive necrosis with broad bands of polymorphonuclear leukocytic infiltration (*see* Fig. 13). The necrosis was in large part complete, involving the interstitial tissue as well as the fibers. The latter were widely separated; some showed discoid degeneration, however, most were swollen and structureless. There was extensive necrosis and thrombosis of both arteries and veins (*see* Fig. 13). In a few of the latter, organization was taking place. In some small areas there was viable tissue; here granulation tissue formation and abortive muscle cell regeneration were occurring.

The flexor hallucis longus appeared grossly normal in color (*see* Fig. 7). Microscopically, it showed extensive necrosis with thin borders of polymorphonuclear leukocytes. The fibers were for the most part minimally to slightly separated from one another. The majority of them showed advanced discoid necrosis; some exhibited variable degrees of swelling and loss of structure. There was considerable fragmentation of fibers (*see* Fig. 9). The muscle nuclei had completely disappeared except in a few areas where they were pyknotic. No congestion, granulation tissue response, or regeneration was seen. Many bacilli were scattered throughout the muscle. The extensor hallucis longus appeared grossly slightly paler than normal (*see* Fig. 7). Microscopically, it showed a picture of necrosis similar to that of the flexor hallucis longus (*see* Fig. 10). Gram-positive rods were seen, especially in the outer portion of the muscle.

Comment. Again, in this case, the gastrocnemius muscle showed less damage than the soleus. The former exhibited a pale-yellow color that corresponded microscopically to extensive early regeneration. Had the limb survived, the amount of regeneration was probably sufficient to have been able to reform a considerable portion of the muscle. The soleus exhibited a characteristic picture of muscle infarction in which pale-brown areas of necrosis alternated with yellowish-white bands of polymorphonuclear exudate. The flexor hallucis longus and extensor hallucis longus showed the most advanced necrosis but the least in the way of color changes. Presumably, the almost complete interruption of blood supply prevented depigmentation of the muscle from occurring.

Case 29. This 25-year-old soldier sustained a gunshot wound which partially severed his left popliteal artery. A tourniquet was in place for 11 hours. On admission, the leg was cold and pale. The artery was anastomosed 16.5 hours after injury; there was no venous involvement; several medium-sized collateral vessels were sacrificed. Postoperatively there were good pulses.

Thirteen days after injury, the anterior and lateral muscles of the leg were excised because of necrosis. The muscles were described as being soft and pale. Microscopically there was extensive advanced necrosis, mostly of the discoid type. In some fragments fiber swelling and hyalinization were prominent. In many areas the dead fibers were closely packed; in others they lay in a sea of exudate. There were wide bands of dead muscle containing large numbers of disintegrating polymorphonuclear leukocytes. Several large areas characterized by marked histiocytic digestion of muscle sarcoplasm, and early regenerative activity were seen. There were fibers in these more viable regions which stained deep blue suggesting calcification.

The leg was amputated above the knee 18 days after injury, and was submitted to the laboratory in formalin. The artery was thrombosed in the region of the anastomosis. The gastrocnemius appeared normal grossly.

Microscopically, it was essentially negative except for slight over-prominence of cross-striations and spotty areas of waxy swelling of fibers. Some of the swollen fibers were being engulfed by histiocytes. The soleus was pale yellow-tan and edematous. Microscopically, the one section taken showed large numbers of very thin regenerating fibers arrayed in an orderly fashion in a very edematous connective tissue stroma (*see* Fig. 19). Between the fibers were small numbers of lymphocytes, plasma cells and histiocytes. The new muscle cells were for the most part elongated and were one-fifth to one-tenth the diameter of normal fibers. Longitudinal fibrils and, in some instances, cross-striations could be identified. The nuclei were oval and were often present in clusters or rows. In a few small areas, groups of intact fibers of normal size remained. The tibialis posterior muscle was very soft and liquefied. Microscopically, fragmented fibers showing discoid necrosis and occasionally loss of structure and swelling were widely separated by blood and disintegrating acute inflammatory exudate. Peripheral to the necrotic areas, the dead muscle had been largely removed so that there remained edematous collagenous tissue containing cavernous blood vessels and acute and chronic inflammatory cells. Here and there were foci of muscle cell regeneration, but this was by no means a prominent feature.

Comment. Again, in this case, there were different degrees of ischemic change in the muscles examined. The anterior and posterior tibial muscles showed the most advanced degrees of necrosis. In the latter, zones of fibrous tissue containing inflammatory cells had

already appeared peripheral to the areas of complete necrosis. The gastrocnemius showed little or no damage. The slight exaggeration of cross-striations was probably of no significance. The soleus, which was pale yellowish-tan and grossly edematous, showed extensive regeneration. The new fibers were numerous and arrayed in an orderly fashion; likewise, there was a dearth of new collagenous tissue formation here. For these reasons, it appears possible that this muscle could have been reformed to a large extent if the limb had remained intact.

Case 30. This 32-year-old soldier sustained a shell-fragment wound involving his right lower thigh. The wound was débrided at a front line hospital. Two days later he developed a massive secondary hemorrhage. The wound was explored and the popliteal artery and vein were found to be severely lacerated. Both were ligated. Gangrene of the foot and lower leg developed and the leg was disarticulated at the knee joint 12 days after wounding. Pathologically, the skin of the leg showed reddish-purple discoloration and zones of desquamation and vesicle formation. The foot was edematous and the tips of the toes were black. The popliteal artery contained a thrombus which extended for a short distance into the anterior tibial artery. The peroneal, posterior tibial, and lower anterior tibial arteries were completely patent. The venae comites were distended by clotted blood. The muscles were described as being pale; and the soleus was said to be pale yellow with focal hemorrhages.

Microscopically, several sections were taken from the gastrocnemius muscle. Two from its mid-portion were essentially negative, although several large veins were greatly distended with thrombi which showed early organization. A third section showed a diffuse area of regeneration in which were scattered small patches of necrosis. The regeneration was characterized by the presence of large numbers of newly formed, elongated muscle fibers separated by edema fluid, large numbers of histiocytes and lymphocytes, and proliferating capillaries and fibroblasts (*see Fig. 20*). The new fibers varied in width from one-fifth to one-third that of normal fibers. Longitudinal fibrils, as well as occasional cross-striations, were perceptible. Nuclei were numerous and were arranged in tightly packed rows or clusters. Here and there were small areas occupied by normal muscle fibers. The patches of necrosis were characterized centrally by complete death of both fibers and interstitial tissue. The former were partly discoid, partly structureless. There were small foci of hemorrhage.

At the periphery was a variable amount of penetration of dead muscle by proliferating capillaries and histiocytes and, to a lesser extent, by fibroblasts and regenerating muscle cells. The medium-sized vessels were congested throughout the section. A large vein was distended with an early organizing thrombus. The corresponding artery was not remarkable. The soleus muscle showed extensive

necrosis. The fibers were mostly swollen and structureless and were slightly to markedly separated by disintegrating polymorphonuclear exudate and edema fluid. The nuclei had disappeared. Histiocytic digestion of dead sarcoplasm and capillary penetration were proceeding to a slight degree at the periphery. The tibialis anterior showed advanced, complete necrosis without congestion or inflammatory cell infiltration. The fibers were minimally to moderately separated; many were structureless; others showed discoid necrosis. Nuclei were severely degenerated. The interstitial tissue was obviously necrotic.

Comment. This case illustrates again the several types of reaction patterns occurring in the muscles of ischemic limbs, ranging from complete necrosis to maintenance of normal structure. There was marked muscle cell activity and only slight fibroblastic proliferation. Had the limb remained intact, it would appear that the end result of such a process might have been an extensively regenerated muscle with at most focal necrosis and slight, diffuse fibrosis.

Case 31. This 23-year-old soldier sustained an accidental gunshot wound which lacerated his left brachial artery. An anastomosis was performed. On arrival at another hospital 6 days later the hand and, to a lesser extent, the forearm were cyanotic. By the 12th day, a line of demarcation appeared 3 inches below the elbow.

An amputation was performed slightly below this level in an attempt to save the joint. On pathologic examination, the hand, wrist, and lower forearm were covered by dry, darkened skin. A short distance distal to the amputation level, the subcutaneous tissue became edematous. The superficial veins were extensively thrombosed. Here and there, the arterial system was filled with blood clots which may have been antemortem in time of formation. Sections taken through both deep and superficial muscles revealed advanced necrosis involving both fibers and interstitial tissue. The fibers were minimally to slightly separated; their cross-striations were exaggerated often to the point of disc formation. In some fibers, the striations were blurred. The nuclei were severely pyknotic. In some areas, the fibers had undergone granular disintegration. There was neither congestion nor inflammatory cell reaction. A sample of muscle taken at the level of amputation showed advanced necrosis.

Thirteen days later, on the 25th day after injury, the limb was re-amputated above the elbow joint. Pathologic examination revealed that the brachial artery was thrombosed at the level of the joint. The muscle in the specimen varied in appearance. In the upper portion, most of it seemed grossly unremarkable. Two types of muscle appeared, especially in the lower regions; one had a buff color and somewhat pasty consistency, the other had a waxy pale-yellow hue (the color of fat) and a normal consistency. Under the microscope, the grossly normal muscle showed focal fiber damage, infiltration of chronic inflammatory cells, and fibrosis, as well as small areas of in-

farction and regeneration. However, the great bulk of the muscle was essentially normal. The pale-yellow, waxy muscle showed massive necrosis of fibers and interstitial tissue. The fibers were slightly to moderately separated from one another.

In the outer areas, exudate composed of fluid, polymorphonuclear leukocytes, and nuclear debris lay between them. Many of them showed advanced discoid necrosis; others exhibited swelling and hyalinization (*see* Fig. 15). The nuclei had almost entirely disappeared. At the periphery, there was marked fibroblastic proliferation and collagen formation (*see* Fig. 15), chronic inflammatory cell reaction, some phagocytosis of dead fibers, and regeneration of muscle nuclei. The latter, though extensive, was limited in its progress by the abundant collagen formation and was, therefore, functionally ineffectual. The section taken from the buff-colored pasty muscle showed the most advanced necrosis. Here many of the fibers had disintegrated to form a fusing granular mass. Most of the fibers were swollen and hyaline; and discoid degeneration was not as prominent as in the previous type of muscle. In several fragments, there was a very thin layer of necrotic exudate at the periphery.

Myoglobin and hemoglobin assays of the normal appearing muscle and the pale-yellow, waxy muscle were performed according to the method of Biorek.¹⁷ The results are presented in Table 3.

Table 3. Pigment Content of Muscle*

Pigment	Normal Muscle		Yellow Muscle	
	First Determination	Second Determination	First Determination	Second Determination
Myoglobin	5. 12	5. 09	2. 57	2. 52
Hemoglobin	. 27	. 26	. 53	. 50

*Given as per cent dry weight (12 hours at 120° C.).

Comments. In this case, the muscles of the original amputated specimen exhibited changes indicating an almost complete interruption of arterial blood supply. The presence of extensive necrosis at the level of the amputation presaged the need for further surgery. The muscles in the second specimen obviously had received a better supply of blood than those in the lower forearm. Some of the former muscles were normal; others showed necrosis, but depigmentation and marked inflammatory and reparative phenomena had also occurred. The yellow waxy muscle was of great interest in that gross depigmentation was accompanied by a marked decrease of myoglobin content.

References

1. Harman, J. H.: A Histological Study of Skeletal Muscle in Acute Ischemia. *Am. J. Path.* 23: 551, 1947.
2. Clark, W. E. LeG.: An Experimental Study of the Regeneration of Mammalian Striped Muscle. *J. Anat.* 80: 24, 1946.
3. Mallory, T. B.: Pathology of the Crush Syndrome in Battle Casualties, p. 272, *The Physiologic Effects of Wounds*. Office of The Surgeon General, Department of the Army, Washington, D. C., 1952.
4. Wilson, W. C.: Occlusion of the Main Artery and Main Vein of a Limb. *Brit. J. Surg.* 20: 393, 1932.
5. Miller, H. H., and Welch, C. S.: Quantative Studies on the Time Factor in Arterial Injuries. *Ann. Surg.* 130: 428, 1949.
6. Harman, J. W.: The Significance of Local Vascular Phenomena in the Production of Ischemic Necrosis in Skeletal Muscle. *Am. J. Path.* 24: 625, 1948.
7. Brooks, B.: Pathologic Changes in Muscle as a Result of Disturbances in Circulation. An Experimental Study of Volkmann's Ischemic Paralysis. *Arch. Surg.* 5: 188, 1922.
8. Barnes, J. M., and Trueta, J.: Arterial Spasm. An Experimental Study. *Brit. J. Surg.* 30: 74, 1942.
9. Lewis, R. B.: Pathogenesis of Muscle Necrosis Due to Experimental Local Cold Injury. *Am. J. Med. Sci.* 222: 300, 1951.
10. Clark, W. E. LeG.: Quoted by J. R. Hughes, Ischemic Necrosis of the Anterior Tibial Muscles Due to Fatigue. *J. Bone and Joint Surg.* 30B: 581, 1948.
11. Montagnani, C. A., and Simeone, F. A.: Observations on the Liberation and Elimination of Myohemoglobin and of Hemoglobin After Release of Muscle Ischemia. *Surgery*, 34: 169, 1953.
12. Bowden, R. E. M., and Gutmann, E.: The Fate of Voluntary Muscle After Vascular Injury in Man. *J. Bone and Joint Surg.* 31B: 356, 1949.
13. Griffiths, D. L.: Volkmann's Ischaemic Contracture. *Brit. J. Surg.* 28: 239, 1940-1941.
14. Bywaters, E. G. L.: Ischemic Muscle Necrosis. *J. A. M. A.* 124: 1103, 1944.
15. Belsey, R.: Discussion on the Effects on the Kidney of Trauma to Parts Other Than the Urinary Tract, Including Crush Syndrome. *Proc. Roy. Soc. Med.* 35: 321, 1941.
16. Blomfield, L. B.: Intramuscular Vascular Patterns in Man. *Proc. Roy. Soc. Med.* 38: 617, 1945.

Chapter 13

The Criteria for Determining the Viability of Muscle in War Wounds

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One of the prime requisites of adequate initial surgery in war wounds is the removal of all devitalized tissue. When all devitalized tissue is removed, delayed primary closure can be carried out in 4 to 6 days. On the other hand, necrosis results if devitalized muscle is allowed to remain in the wound. Necrotic tissue serves as a stimulus to bacterial growth with subsequent local infection, septicemia, or clostridial myositis; and in some instances it causes death.

Determination of the viability of muscle soon after wounding is difficult. Thorough débridement in large wounds is a real problem even for the most experienced wound surgeon. A surgeon who has had considerable experience in débriding wounds has developed sufficient clinical judgment to accomplish a good débridement. The exact criteria used vary with the surgeon, as they are based on individual experiences. Some surgeons determine viability at operation primarily by contractility of the muscle, some place greater emphasis on the ability of the muscle to bleed, while others judge the condition of the muscle by its color and consistency.

It appears that there are four criteria—color, consistency, contractility, and bleeding—which may be used to determine the viability of muscle. This study was initiated in an attempt to evaluate the importance of each of these criteria.

Materials and Methods

Sixty biopsies of muscle of questionable viability were taken from war wounds of various regions of the body by two surgeons at the time of initial débridement. The average time from injury to surgery was 6 hours. The muscle was observed carefully before biopsy and graded as to color, consistency, contractility, and bleeding. Whenever possible, both surgeons corroborated in grading the specimen. Observations made for color were: dark red, red, pale, or pink. Pink was used as the color of normal, noninjured muscle. Gradations

of consistency were mushy, stringy, soft, and firm; and they were determined by grasping the tissue with forceps and observing the character of the muscle. Sometimes it was difficult to grade the amount of contractility. The muscle mass was pinched with a forceps and its reaction observed and graded 0, 1+, 2+, or 3+. Normal muscle bleeding was graded as 3+. The biopsies were put in a saline solution for 20 minutes, then fixed in 10 per cent formalin. The pathologist received each specimen with a clinical note stating its gradation as to color, consistency, contractility, and bleeding.

The individual biopsies varied from 1–2 cm. in diameter. Each one was examined following fixation by the pathologist; and it was cut with a sharp scalpel blade if trimming was necessary. The tissues were carried through paraffin embedding and were stained with hematoxylin and eosin. The pathologist examined and interpreted the specimens independently of the surgeon's description of the muscle. Each biopsy was graded according to an estimation of the percentage of damaged muscle in the total specimen, with ranges as follows: none to minimal damage, 0–10 per cent; slight damage, 11–33 per cent; moderate damage, 34–66 per cent; marked damage, 67–89 per cent; complete or almost complete damage, 90–100 per cent.

Results

Miscroscopic Findings

The qualitative degree of damage in the débrided muscle exhibited a wide range. Because of the fine shading of the various types of damage into one another, it was decided to grade the damage quantitatively and not qualitatively. The least evidence of muscle injury was separation of individual fibers from one another. Since this finding is a common artefact in tiny fragments and along the borders of large fragments, it was the most difficult to evaluate and was therefore interpreted cautiously. Fortunately only a few specimens fell into this category.

A muscle injury considered somewhat severe was the separation of fibers and degenerative changes in individual fibers manifested by nuclear pyknosis and various alterations in the sarcoplasm. These alterations included: (1) waxy degeneration, indicated by fibers which had taken on a homogeneous, deeply eosinophilic hue, and (2) sarcoplasmic shredding, manifested by the sarcoplasm being contracted into coarse reticular masses so that the remainder of the sarcolemmal tubes were filled with slightly basophilic fluid.

The last two forms of muscle degeneration offered difficulties in interpretation, since they were occasionally present as artefacts at the cut edges of the muscle fragments. The discoid type of degeneration which is observed in ischemic muscle was not seen in these me-

chanically damaged muscles. The most advanced changes noted in the injured muscle were marked blurring of the internal structure of the fibers, with advanced pyknosis, and disappearance of nuclei. These fibers were most often ballooned into swollen masses widely separated from one another. The endomysial framework of such muscle was frequently disrupted, with a disorderly arrangement of the fragmented fibers. This advanced form of degeneration undoubtedly represents irreversible necrosis.

Other findings in the biopsies were edema, polymorphonuclear leukocyte infiltration and hemorrhage. The presence and distinctness of the cross-striations were of little help in judging the degree of muscle damage. Although the striations were seldom recognizable in the most advanced forms of necrosis, frequently they were sharper and more clearly visible in obviously damaged muscle than in apparently normal muscle. These changes tended to be most marked in the perimysium, and they appeared to a lesser extent in the endomysium. They were rarely diffuse, although occasionally they were intense focally. Their presence alone was not considered indicative of muscle degeneration.

No observations were made of intravascular accumulations that could be identified with certainty as thrombi; however, it is probable that many of the intravascular red-cell clumps were actually thrombotic. The vessels, especially the veins, often showed striking polymorphonuclear infiltrations of their walls. Another finding of interest was the structural replacement of large portions of disrupted vascular wall by coagulums of fibrin, platelets, and a few cells.

Surgical Pathologic Correlation

Table 1 presents the over-all correlation of surgical criteria for muscle damage, with the amount of damage observed microscopically. Plotted from Table 2, the probit shows excellent linearity (Fig. 1). Thus, the population of the specimens was a statistically normal one. This finding adds weight to the reliability of the results.

Correlation coefficients were computed for the relationship between the various surgical criteria in each category: color, consistency, ability to bleed, contractility, and the amount of muscle damage observed microscopically. These coefficients are shown in Table 3. The best correlation between the surgical criteria and the amount of damage observed microscopically by the pathologist was found in the consistency category. However, the correlation was almost as significant in the categories of ability to bleed and contractility. Correlation between color changes and the amount of microscopic damage was doubtful from the standpoint of statistical evaluation of the data.

Table 1. Correlation of Surgical Criteria with Damage Observed Microscopically

Degree of Damage	Number of Cases	Color				Consistency				Ability to Bleed				Contractility*			
		Dark Red	Red	Pale	Pink	Mushy	Stringy	Soft	Firm	0	1+	2+	3+	0	1+	2+	3+
Minimal 0-10%	7	2	5	0	0	1	2	0	4	2	3	0	2	4	1	1	1
Slight 10-33%	11	5	4	1	1	0	0	7	4	3	3	5	0	7	1	2	1
Moderate 34-66%	20	14	5	0	1	3	1	8	8	5	9	5	1	9	7	2	2
Marked 67-89%	10	5	4	0	1	3	3	3	1	6	3	1	0	9	1	0	0
Complete 90-100%	12	8	4	0	0	7	4	1	0	9	3	0	0	12	0	0	0
Total	60	34	22	1	3	14	10	19	17	25	21	11	3	41	10	5	4

*Degree of response: 3+, normal; 2+, less than normal; 1+, minimal; 0, none.

Criteria for Determining Muscle Viability

Table 2. Computation of Probit Line Plotted from Data in Table 1

Degree of Damage	Number of Cases	Cumulative Cases	Cumulative %* ($\Sigma f - V_2$)	Probit*
Minimal	7	7	10.8	3.762
Slight	11	18	29.1	4.450
Moderate	20	38	62.5	5.319
Marked	10	48	79.1	5.810
Complete	12	60	99.1	7.376

*From Moore, F. J., Cramer, F. B., and Knowles, R. G.: *Statistics for Medical Students and Investigators in the Clinical and Biological Sciences*. The Blakiston Company, Philadelphia, 1951.

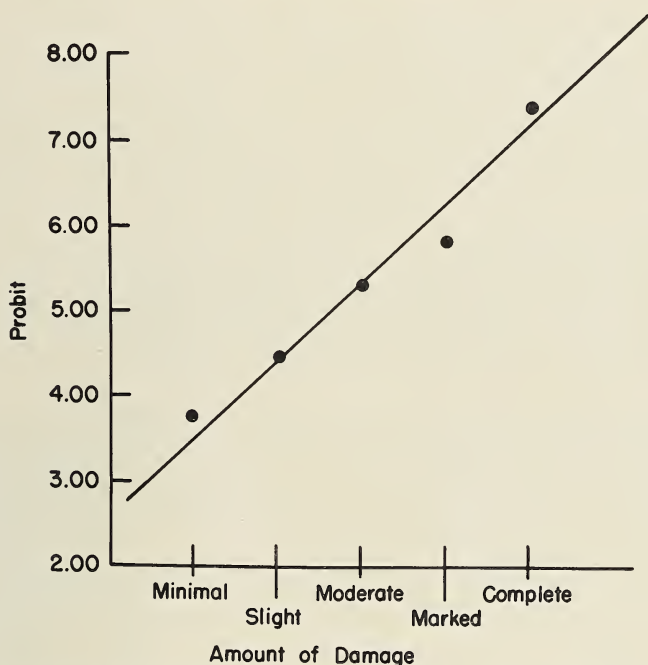


FIGURE 1. Probit line plotted from data in Table 2. The linearity shown here points out the normality of the population of specimens that were examined.

In general, the studies showed that there are three valid criteria for determining the viability of muscle: consistency, ability to bleed, and contractility.

Discussion

There are several obvious defects inherent in using pathologic criteria to determine muscle viability. It is possible that muscle, which appears normal on routine staining with hematoxylin and eosin,

Table 3. Correlation Coefficients from Analysis of Data in Table 1

Criterion*	Correlation Coefficient† (~)	P Value
Consistency	-0.529	<.001 (very significant)
Ability to bleed	-0.418	<.001 (very significant)
Contractility	-0.353	0.01 > P > .001 (significant)
Color	-0.230	~.05 (borderline)

*Number of cases, 60; number of degrees of freedom, 58.

†Computed according to Snedecor, G. W.: *Statistical Methods*, 4th edition, p. 138. Iowa State College Press, Ames, Iowa, 1946.

may actually be in the initial stages of irreversible necrosis. Moreover, muscle showing minor to moderate degrees of regeneration probably is capable of restoration to normal. Despite these possibilities, it is logical to assume that the actual degree of degeneration and necrosis, in general, parallels the microscopically observed severity of damage, and that morphologic changes are valid criteria for comparative evaluation of viability.

The biopsies in this series were taken from 3 to 8 hours after wounding. The time factor did not seem important, as the muscle specimens taken 3 hours after wounding were capable of showing the same grades of clinical and pathologic damage as those taken at 8 hours.

A second defect inherent in the method is the difficulty in quantitating selectively the various qualitative types of muscle damage observed. The biopsies showed degrees of damage ranging from mild to severe. Although the damage most often involved diffuse areas, occasionally it was manifest as focal fiber damage.

A third clinical difficulty is the variation of terms used by individuals to express the surgically observed characteristics of a small section of muscle. For this reason, any results of such a study must be interpreted by each surgeon in the light of his own personal experiences in débriding wounds. A further refinement in this type of evaluation would be a careful clinical follow-up of the wounds by the surgeon, possibly with additional biopsies made on an area near that of the original ones. This procedure would give an indication as to the ultimate outcome of the muscle tissue in that area.

It is well recognized that it is most difficult for the surgeon to make a decision as to the viability of muscle, and it requires considerable experience. Most surgeons gain insight into the problem by re-evaluating the efficiency of their débridement upon examination of the wound 4 or 5 days after the initial operation. Judgment improves

after several experiences of this type; and a better initial débridement is carried out.

Although the results of this study are limited, they indicate that there is a statistically significant correlation between consistency, ability to bleed, contractility, and the amount of damage that is observed microscopically. There is sufficient evidence to show that a muscle is viable if it is firm, if it bleeds when cut, and if it contracts when stimulated.

Summary and Conclusions

Preliminary study was carried out to observe the criteria for the viability of muscle in wounds requiring débridement.

Sixty biopsies were taken from 12 war wounds from 3 to 8 hours after injury. Each portion of muscle observed was graded by two surgeons according to color, consistency, ability to bleed, and contractility. The pathologist graded each specimen independently to determine the amount of necrosis: minimal, slight, moderate, marked or complete. These biopsies were taken from areas of questionable necrosis; and they were estimated quantitatively only and then correlated with these various surgical criteria.

A statistically significant correlation was obtained for three criteria: consistency, ability to bleed, and contractility. Color was of doubtful value in determining the viability of muscle.

Chapter 14

Field Management of Burns

Lieutenant Colonel Curtis P. Artz, MC, USA

During the period from February 13, 1953, to August 1, 1953, seven burned patients were studied at the 46th Surgical Hospital in Korea. Several burned patients were seen in other hospitals; and conferences were held with various medical officers in the theater concerning their experiences in the management of burns in the field. Two conferences were held with the physicians in charge of the Burn Ward at the Tokyo Army Hospital.

Of the seven burned patients treated at the 46th Surgical Hospital, two had 30 per cent full-thickness injury, with 5 or more per cent partial-thickness burn. Both patients with full-thickness burns were injured from gas stove explosions. With the exception of the face, which was exposed, these patients were treated with the Universal Protective Dressing. They received initial fluid replacement therapy at the 46th Surgical Hospital and were in good condition when evacuated. On the eighth day following injury, one of these patients was admitted to the Burn Study Ward at Brooke Army Hospital; his initial dressings and experimental hand splints were in place. The other patient was admitted to Brooke Army Hospital on the 11th day following injury; but all of his dressings had been changed except those on his hands where experimental hand splints were in place. Follow-up study on these two patients showed that they withstood evacuation to the United States quite well, and the experimental splints had held the hands in good position.

When one patient accidentally stepped in front of a flame thrower, he experienced a 95 per cent, full-thickness burn. He expired 8 hours after admission to the hospital.

Four patients sustained 10 to 15 per cent full-thickness burns of various parts of the body. Comparison of the use of the exposure method and the Universal Protective Dressing showed that healing occurred at the same rate in these patients. However, patients in the field hospital stated that the dressed areas were more comfortable than the exposed areas. These patients were treated during the late winter months, and they complained of a moderate amount of cold and discomfort in the exposed areas. It was observed that these patients could be moved more easily when dressings were in place. On the other hand, good results were obtained by the exposure method; and this type of therapy is feasible in a field hospital.

The Universal Protective Dressing proved to be of considerable value. On July 21, 1953, an evaluation report on its use was sent to the Surgeon of the 8th United States Army. Various items in this report are listed below.

1. During the period from February 1953 to July 1953, the 46th Surgical Hospital used 100 Universal Protective Dressings.

2. A few of these 100 dressings were used on five patients who had suffered severe burns; and the others were used on patients with large wounds of the extremities.

3. Several follow-up questionnaires were completed and inserted into the patients' charts at the time of evacuation.

4. It was observed that the Universal Protective Dressing was most efficacious in the management of burns. It did not soak through for several days. It was pliable and could be molded to the affected part. The thickness of the present dressing appeared to be adequate. A thicker dressing would be less desirable because it would be more difficult to apply evenly; and it could not be used to exert an even pressure. The two sizes in use at present appear to be satisfactory (22 x 36 inches, and 22 x 16 inches).

5. Most of the dressings were applied over large wounds of the extremities or over multiple wounds of the extremities. A great deal of time is saved in the application of this type of dressing. It is absorptive and serves to splint the affected part. It proved to be useful in the early management of wounds in a forward surgical unit.

6. Packaging of the Universal Protective Dressing is satisfactory. However, the roll of bandage is not satisfactory because it has insufficient elasticity. A chemically crinkled bandage would be desirable. The package should also include four rolls of bandage with the 22 x 36 inch dressing.

7. Universal Protective Dressings are of great advantage, since they afford considerable saving of time. They should be made available to all medical installations in forward areas.

Prior to their admission to the surgical hospital, all burned patients had had their wounds débrided. In most instances, the wounds had been washed with soap and water and some type of covering had been applied. This treatment appeared to be satisfactory, since the burned surfaces had been cleansed thoroughly, devitalized tissue had been removed, and dressings had been applied to wounded areas. As evacuation time in the case of most burned patients was between 2 and 4 hours, it appears that some type of clean covering is necessary before a patient is transported to an installation where more definitive therapy may be carried out. It is imperative that original dressings be removed in order that physicians who are responsible for replacement therapy can estimate the severity of injuries.

Suggestions for the Management of Burns in the Field

At the Battalion Aid Station

The patient suffering from discomfort should be given morphine intravenously; and he should also be given dextran. If dextran is not available, saline solution should be started. The burned area should be covered with some type of dry, sterile dressing if it is available. For this initial cover of the injured area, a Universal Protective Dressing is ideal, since it affords a good splint during the period of transportation. It is unnecessary to cover burns of the face. A burned patient should be evacuated to a clearing station or a surgical hospital. At a clearing station, all burned patients should receive fluid replacement therapy and definitive local care; then they should be removed to an evacuation hospital 48 hours later. If the chain of evacuation is from the clearing station to the surgical hospital and thence to the evacuation hospital, it would seem wise to perform definitive local care and fluid replacement therapy at the surgical hospital.

At the Clearing Station or Surgical Hospital

All dressings should be removed as soon as the patient arrives, and the general status of the patient and extent of injury should be evaluated. Fluid replacement therapy should take precedence over local care. A catheter should be placed in the bladder and a cannula inserted in a vein in patients whose burns involve more than 20 per cent of their body surface.

Replacement Therapy. Fluid replacement therapy should be estimated in accordance with the percentage of body surface burned and the size of the patient. Adequacy of this therapy and rate of administration of fluids should be governed by the output of urine which should be kept at from 30 to 50 cc. per hour. Fluid therapy may be estimated by the method outlined in Tables 1 and 2.

In general, the colloid requirements can be met in large, full-thickness burns with a ratio of 1 unit (500 cc.) of dextran to 2 or 3 units of blood. In partial-thickness burns, dextran should be used primarily for colloid replacement.

A rapid, easy method of estimating the amount of fluid required is by the use of the fluid calculator prepared by the Surgical Research Unit at Brooke Army Medical Center (Fig. 1). This calculator should be available in all clearing stations and surgical hospitals.

Patients who have sustained burns of less than 20 per cent of their body surface may be resuscitated with oral fluids. A well refrigerated, salt-soda solution containing 3 gm. of sodium chloride and 1.5 gm. of sodium bicarbonate per liter is well tolerated. This solution should be administered for 2 days.

Table 1. Fluid Replacement Therapy in the First Twenty-four Hours

Solutions	Formula	Example
Colloid: Blood or Plasma or Plasma expanders	$0.5 \text{ cc.} \times \text{body weight (kg.)} \times \text{per cent of body surface burned}$	155 lbs. (70 kg.) man with 30% burn of body surface: $0.5 \times 70 \times 30 = 1,050 \text{ cc.}$
Electrolyte: Lactated Ringer's or Saline	$1.5 \text{ cc.} \times \text{body weight (kg.)} \times \text{per cent of body surface burned}$ ($3 \times \text{above amount}$)	for the same patient: 3,500 cc.
Glucose in water or Lactated Ringer's	use a fixed amount, <i>i. e.</i> , 2,000 cc.	total in example = 6,200 cc.
		Give $\frac{1}{2}$ in first 8 hours Total = 3,100 cc.
		Give $\frac{1}{4}$ in next 8 hours Total = 1,550 cc.
		Give $\frac{1}{4}$ in next 8 hours Total = 1,550 cc.

Table 2. Fluid Replacement Therapy in the Second Twenty-four Hours

Solutions	Formula	Example
Colloid	$\frac{1}{2}$ to $\frac{3}{4}$ of the above amount	for the same patient: $\frac{1}{2} \times 1,050 \text{ cc.} = 525 \text{ cc.}$
Electrolyte	$\frac{1}{2}$ to $\frac{3}{4}$ of the above amount	for the same patient: $\frac{1}{2} \times 3,500 \text{ cc.} = 1,750 \text{ cc.}$
Glucose in water or Lactated Ringer's	use a fixed amount, <i>i. e.</i> , 2,000 cc.	total in example = 4,275 cc.
		Give throughout the day

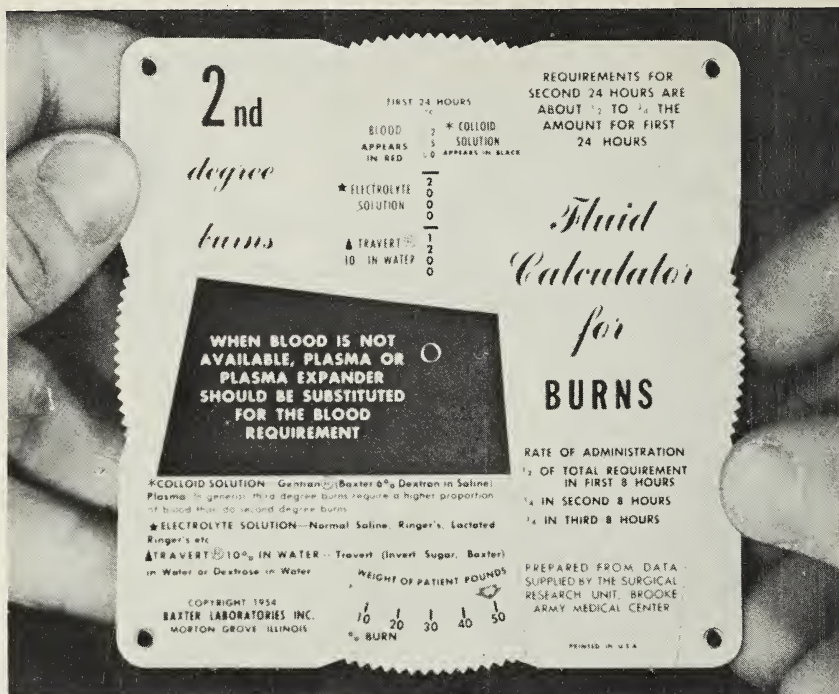


FIGURE 1. A pocket-sized, plastic, fluid calculator for burns. The opposite side shows the requirements for 3d-degree burns.

Local Care of the Burn Wound. As soon as fluid replacement therapy is assured, the patient should be taken into the operating room and, under aseptic conditions, the burned surfaces should be cleansed with soap and rinsed with copious amounts of water. All blisters should be broken, debris removed, and detached epithelium cut away. No local applications of any sort should be used. If permitted to remain exposed, the face, neck, and upper chest heal well. The patient appears to be more comfortable when these areas are exposed. All other areas should be dressed with the Universal Protective Dressing. In the event that these dressings are not available, all areas may be exposed.

It is important that the hands be placed in a position of function; and they should be bandaged with a large occlusive dressing. The dressings should remain in place for approximately 10 days, unless there is undue pain, fever, saturation, or other evidence of infection. Always give tetanus toxoid. Systemic penicillin should be administered in large doses for a minimum of 5 days, and thereafter antibiotics given on specific indications. It is wise to perform a tracheotomy in the case of full-thickness burns of the face. If the eyelids are burned deeply, a tarsorrhaphy should be performed (between the

5th and 10th day) ; otherwise ectropion will develop with subsequent drying and ulceration of the cornea.

A tracheotomy must be performed when respiratory tract involvement is suspected; and fluids must be administered cautiously thereafter to avoid pulmonary edema.

Usually initial cleansing is performed on the freshly burned surface under intravenous morphine analgesia. If a patient is received after development of inflammation and sensory nerve irritation, it may be necessary to add minimum amounts of sodium pentothal, and 50 per cent nitrous oxide with 50 per cent oxygen for basal narcosis.

Other Aspects of Management of Burns

All patients, except those with very minor burns, should be evacuated on about the third day to an evacuation hospital providing replacement therapy has been assured. Patients with minor burn wounds may be treated at the battalion aid station or clearing station and then held for a few days until healing is complete.

At the evacuation hospital, patients should be further evaluated at the time of their first change of dressing. Further movement rearward to a general hospital in the Communications Zone is dependent upon the local policy of evacuation.

It seems desirable to designate as burn centers certain evacuation and Communications Zone hospitals, as well as hospitals in the Zone of Interior. Patients with this type of injury receive better care when they are treated by a specially trained team who are experienced in the care of burns.

References

1. Artz, C. P.; Reiss, E.; Davis, J. H.; and Ampsacher, W. H.: The Exposure Treatment of Burns. *Ann. Surg.* 137: 456, 1953.
2. Reiss, E.; Stirman, J. A.; Artz, C. P.; Davis, J. H.; and Ampsacher, W. H.: Fluid and Electrolyte Balance in Burns. *J. A. M. A.* 152: 1309, 1953.
3. Reiss, Eric, and Artz, C. P.: Current Status of Research in the Treatment of Burns. *Mil. Surg.* 114: 187, 1954.
4. Artz, Curtis P., and Reiss, E.: Calculator for Estimating Early Fluid Requirements in Burns. *J. A. M. A.* 155: 1156, 1954.

Chapter 15

Clostridial Myositis—Gas Gangrene:

Observations of Battle Casualties in Korea*

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The period, January 1952 to June 1953, was a time when the front lines on the eastern front in Korea were stable. Only an occasional casualty failed to reach a battalion aid station within a few hours following injury. The flow of casualties was usually light, so that most patients could receive immediate attention at the battalion aid station and throughout the forward chain of evacuation. Each patient was given a booster injection of tetanus toxoid and 300,000 units of penicillin, as well as other resuscitative measures. He was then evacuated to the 46th Surgical Hospital (Mobile Army) which was located approximately 10 miles behind the front lines.

During this phase of the war, the average period of evacuation from the time of wounding to the time of reaching an American hospital in this sector was only 3.5 hours. Furthermore, because of the relatively light flow of casualties, the preoperative time-lag at the hospital was usually negligible. Early, definitive surgery was therefore possible. Primary débridement and secondary closure constituted a standard policy for the management of most wounds. Penicillin was used routinely during the postoperative period. Streptomycin and occasionally Aureomycin were also used following visceral perforations or vascular injuries.

Patients were held at a forward hospital from 1 to 7 days prior to evacuation. Whereas evacuation from a battalion aid station had been by ambulance or helicopter, evacuation from a forward hospital to an evacuation hospital was entirely by air. All casualties, except neurosurgical patients, were evacuated from the forward hospital to the 11th Evacuation Hospital located approximately 90 miles behind the front lines. Patients were held at this secondary installation until ready for prolonged trips by train or air to hospitals further to the south or in Japan. Severely injured men were usually held at one of these two hospitals for a minimal period of 7 to 10 days.

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During the 18 months of this study, 4,900 battle casualties were observed on the general surgical service of the 46th Surgical Hospital. Of the 4,900 patients, only four were recognized as having gas gangrene; and none of these four patients died. Including these 4,900 patients, approximately 12,000 casualties were seen by the staff of the 11th Evacuation Hospital. None of the original 4,900 patients were recognized as having gas gangrene at the secondary installation; and only four of the additional 7,100 patients coming through the evacuation hospital were noted to have or to have had gas gangrene. The latter observation is doubtlessly based on incomplete records. Excluding patients with posttraumatic renal insufficiency, because their wound surgery was often compromised because of their critical condition, only eight patients with gas gangrene or severe clostridial myositis were seen by the authors during 18 months in Korea. None of the eight patients died of the clostridial infection. The only fatalities from clostridial infection seen during this period of time were one or more patients with coincident anuria at the Renal Insufficiency Center. Such a casualty was sometimes in too critical a condition to permit primary or adequate secondary débridement of massive, ischemic wounds.

All of the serious clostridial infections were seen in wounds in which débridement had not been achieved. Most of them were secondary to inadequate arterial repairs in which secondary vascular occlusion to the development of an ischemic, gangrenous limb even though the missile tract was débrided. Thus, a major artery was divided in six patients; an arterial tourniquet was left in place on the seventh patient for 48 hours until his condition improved enough to permit operation; and the eighth patient was in such a critical condition that the severe wounds of the extremities were not explored. In the last patient, interruption of the arterial continuity had doubtless occurred but was not proved.

Perhaps the progress since World War I can best be appreciated by noting an excerpt from the report of Pettit of his experiences in evacuation hospitals during the St. Mihiel and Argonne-Meuse operations.

“Of 890 wounds examined bacteriologically, 478, or 53 per cent, were found to contain anaerobic bacilli. Of these 478 wounds, 321, or 67 per cent, at no time showed clinical evidence of gas infection. Of the remainder (33 per cent), 3 per cent developed gas gangrene after operation—29 per cent had gas gangrene clinically evident at the time the bacteriologic examination was made.”³

Of the 4,377 patients reported by Pettit, 221 (5.0 per cent) developed gas gangrene. The resulting case fatality rate from gas gangrene was 27.6 per cent. This may not be an entirely representative figure from

World War I, for these 4,377 battle casualties were seen between September 10 and November 13, 1918. At this time, a lag period of 48 hours before surgery was not infrequent.

Recording the history of World War I, Coupal wrote: "Of the 224,080 officers and men of our Army who were wounded in battle in France, 13,691 died as a result of their wounds, the total death rate therefore being 6.11 per cent. The wounds complicated by gas gangrene may be divided among those who sustained injuries of the soft parts only and those whose wounds were complicated by bone fracture. Injuries to the soft parts as recorded here do not include those due to chemical-warfare gases. There were, with these exceptions, 128,265 wounds of the soft parts with 9,719 deaths; of the wounded in this group, 1,389 developed gas gangrene, which amounts to only a little more than 1 per cent (1.08). The death rate among those who received wounds of the soft parts which became complicated with gas gangrene was 48.52 per cent, the actual number of deaths being 674.

"Among the 25,272 whose wounds included bone fracture, there were 2,751 deaths. The incidence of gas gangrene among the bone-fracture cases was much higher than among those who sustained wounds of the soft parts only, the total being 1,329 with 593 deaths. The incidence in this group of the wounded was therefore 5.26 per cent and the case mortality rate 44.62 per cent." ⁴

Writing from an American hospital in India during World War II, North described an incidence of clostridial myositis of 0.7 per cent in 4,600 consecutive casualties. The case fatality rate in the 32 patients involved was 31.3 per cent. ⁵

The 4,900 casualties at the 46th Surgical Hospital in Korea offer a reasonable comparison (Table 1). Doubtlessly fewer of the less severely injured casualties by-passed the surgical hospital in Korea than by-passed the evacuation hospital in World War I, but the comparison otherwise would appear reasonable. The soil in Korea, without exception, was found to contain *Clostridium perfringens*, ¹ and the incidence of wound contamination with clostridia approached that found in the wounds of World War I ³ and World War II. ⁶ The incidence of only 0.08 per cent of the clostridial myositis in Korea and the mortality of zero in this series should be attributed to *early, adequate débridement*, to success in the restoration of arterial continuity following injury, and to the early and consistent use of antibiotics. Antitoxins were not used. Tetanus was not recognized in any casualty.

Studies made in collaboration with the Department of Bacteriology of the 406th Medical General Laboratory of the bacterial flora of wounds at the time of primary débridement demonstrated at 41 (26.6 per cent) of 154 wounds contained clostridia which were pathogenic

as demonstrated by guinea pig inoculation. Many of the wounds contained *Cl. perfringens*, yet only one of this entire group developed gas gangrene. Many of the patients had a mild elevation in temperature and pulse, suggestive of a mild systemic reaction to wound infection.¹ Such wounds characteristically contained a mixed culture until the time of secondary closure.² If small areas of necrotic tissue were left, the local infection and systemic reaction were more pronounced. *Cl. perfringens*, as well as other pathogenic clostridia, were present in many of these wounds, and there is no reason to believe that they did not contribute to the toxicity of the wound.

Table 1. Clostridial Myositis (Gas Gangrene)

Conflict	Number of Cases	Number Developing Cl. Myositis	Percentage Developing Cl. Myositis	Case Fatality Rate
World War I ³	4, 377	221	5. 0	27. 6%
World War II ⁵	4, 600	32*	0. 7	31. 3%
Korean Conflict†	4, 900	4	0. 08	0

*An additional 37 patients developed serious clostridial wound infections, making a total incidence of 1.5 per cent of serious infection by *Clostridium perfringens*.

†Emphasis is placed upon the fact that this study was made during the last half of the Korean conflict.

Thus, many of the wound infections were, in part, clostridial infections. The fact that they were usually mild, well tolerated by the patient, and not recognized by the clinician as having a clostridial component should not obscure the underlying process. In this limited sense, clostridial infections were frequent. The term is used here to denote a subclinical clostridial infection and not the wound infection containing gas, which was recognized previously and discussed in the literature. Although clostridial infections were frequent in this limited sense, they were a rarity in the usual sense of life-endangering infections.

Summary

With an average evacuation time of 3.5 hours and a very short time-lag before surgery, severe clostridial infections were rare during the latter half of the Korean conflict. Only four among 4,900 consecutive patients seen on a general surgical service at a forward surgical hospital in Korea during the period from January 1952 to August 1952 were recognized as having clostridial myositis. None of the four died.

The immediate response of an established clostridial infection to adequate surgery and to the continued use of antibiotics led to the conclusion that severe clostridial infections were not a major problem under the existing tactical conditions. All of the prerequisites for life-endangering clostridial infections remained, however, and would doubtlessly become manifest should the time-lag before surgery be again greatly extended.

Case Reports

Patient 1. This 25-year-old American soldier was wounded on September 23, 1952, by artillery-shell fragments. He was admitted to the 46th Surgical Hospital a few hours later. His injuries included an open, comminuted fracture of the lower femur with severance of the popliteal artery. The artery was débrided but re-anastomosed under moderate tension. The distal pulsation subsequently disappeared, although the foot remained warm. On the sixth postoperative day, the patient developed hemoptysis. A diagnosis of pulmonary embolus was made. On the eighth postoperative day, his blood pressure dropped to 70/50; pulse rate increased to 140; and the respiratory rate rose to 54 per minute. He remained afebrile. Although the foot remained warm, gas and edema were evident in the thigh. Exploration of the wound revealed necrotic muscle, obliteration of the arterial continuity, and obvious gas gangrene. A mid-thigh amputation was performed. Four hours following the operation he appeared completely nontoxic.

Penicillin (one million units) and streptomycin (1.0 gm.) were used daily throughout his course. Aureomycin (1.0 gm.) was added after the clostridial infection was recognized.

The patient was evacuated on the 18th day following injury. His convalescence after amputation was smooth. Wound cultures revealed *Clostridium sporogenes*, *sordelli*, *aerofetidum*, and *tetani*.

Patient 2. This 21-year-old Turkish soldier was admitted to another hospital on October 23, 1952, with a wound of the popliteal artery. The arterial involvement was not recognized and the wound was débrided and closed primarily.

Two days later, he was transferred to the 46th Surgical Hospital. The leg from the knee distally was gangrenous. Gas and edema extended proximally into the thigh. The patient appeared to be quite toxic. Under general anesthesia, he developed a mild shock which responded to multiple transfusions. A low thigh amputation was performed. His subsequent course was smooth on two million units of penicillin and two grams of streptomycin daily.

Wound cultures at the time of amputation demonstrated *Cl. perfringens* and *sporogenes*.

Patient 3. This 21-year-old American soldier was wounded at 1300 hours on November 16, 1952, by the explosion of a land mine. His injuries included incomplete amputation of both lower extremities.

He was brought immediately to the forward hospital. His blood pressure was unobtainable. His peripheral pulse and apical heart sounds were imperceptible. The only sign of life was an occasional gasping respiration. Tourniquets were applied and 9 pints of blood were forced intra-arterially within 35 minutes. His blood pressure was then 120/80, pulse rate 127 per minute.

After 2 hours and the infusion of 16 pints of blood, he was taken to the operating room. Without additional trauma beyond this passive movement, his blood pressure again became unobtainable. Meanwhile, under local anesthesia and with the tourniquets still in place, the dangling legs were trimmed off at the knee and a pressure dressing was applied. One mg. of neosynepherine was given by rapid intravenous drip and the pressure showed a transient rise to 100/70. After receiving the 30th pint of blood, his blood pressure rose to 100/80; pulse was 150 per minute. At 1830 hours, which was 5.5 hours following injury, his blood pressure rose to 110/80. It was maintained at approximately this level by the addition of 5 mg. neosynepherine to each bottle. With the tourniquets still in place, he was returned to the ward to await further stabilization before definitive operation was undertaken. For the next two days his blood pressure ranged around 110/70, pulse rate 130, respiratory rate 30. The tourniquets remained on throughout this period and he developed extensive gas gangrene distal to the tourniquets.

On November 19, 3 days after injury, a bilateral amputation was carried out above the tourniquet. The procedure was performed under light nitrous oxide, oxygen, ether anesthesia. Six pints of blood were given (totaling 42 pints), and the 1.5-hour operation was tolerated without a drop in blood pressure. His subsequent course was smooth. His wound culture revealed *Cl. perfringens*.

Patient 4. This American infantryman was admitted to the 46th Surgical Hospital with bilateral, shattering fractures of the femurs due to artillery-shell fragments. Amputations were delayed in the hope of tissue survival and because of the patient's severe hypotensive condition.

His shock was reversed by massive transfusion; but he developed a mild posttraumatic renal insufficiency. His course was febrile, and on the third postoperative day a diagnosis of bilateral gas gangrene was made. A bilateral, high thigh amputation was performed. He almost died of the operative shock, complicated by a severe potassium intoxication. The shock responded to further transfusions and the potassium intoxication responded to dialysis on an artificial kidney. His subsequent improvement was dramatic; and he was evacuated 10 days later.

Wound cultures revealed *Cl. perfringens*, *sporogenes*, *paraputrificum*, and *bifermantans*. Penicillin (one million units) and streptomycin (1.0 gm.) were administered throughout the period of infection.

Patient 5. This American infantryman was admitted to a forward hospital on November 9, 1952. His injuries caused by shell fragments, included a fracture of the femur and a perforation of the popliteal artery. His arterial repair was performed 16 hours after injury.

On the third day following injury, gangrene of the leg was obvious as was crepitation of the thigh. The patient was pale and anorexic. His oral temperature was 98° F. and his pulse rate was 100 to 120 per minute.

With the induction of ether anesthesia, he developed a profound hypotension but recovered as blood was forcibly infused intravenously. Hemorrhagic edema fluid was found extending from the popliteal area upward to the inguinal ligament. The arterial anastomosis was thrombosed.

The facial planes were widely exposed and a mid-thigh amputation was performed. Penicillin and streptomycin therapy had been started on admission and it was continued. His course was exceedingly smooth. Within 10 hours after operation, his blood pressure, pulse, and temperature were almost normal; and he was hungry. Wound cultures included pathogenic *Cl. sporogenes* and *novyi*.

Patient 6. This 22-year-old American soldier was wounded by shell fragments at 1100 hours on February 9, 1953. The popliteal artery and vein were severed; and there was a massive injury to the gastrocnemius. A radical débridement was performed, but tissue of questionable viability was left in the wound. The repair of the popliteal artery was completed at 2300 hours.

Three days later the patient was pale, nauseated, and lethargic. His pulse rate was 120 per minute, and he was afebrile.

The wound was redressed under general anesthesia. The anastomosis was unquestionably patent; but the gastrocnemius and soleus were nonviable. Arterial blood spurted from the cut surface of the dead muscle. A diagnosis of early clostridial infection was made and a supracondylar amputation was performed. His subsequent course was uneventful. Wound cultures demonstrated *Cl. perfringens*.

Patient 7. This American soldier was admitted to the 46th Surgical Hospital a few hours after shell fragments had caused fractures of both bones of the leg and severance of both anterior and posterior tibial arteries. At the time of primary débridement, the foot appeared markedly ischemic but viable.

Because of the clinical evidence of toxicity, the wound was dressed on the third and fourth days after injury. Dressing on the fourth day showed evidence that the tissues contained gas and wine-colored

edematous fluid. Immediate amputation was followed by relief of his systemic reaction. Penicillin and streptomycin were administered throughout his hospital course. Wound cultures demonstrated *Cl. perfringens*.

Patient 8. This American soldier was admitted to a forward hospital with a large penetrating wound of the thigh. Exploration revealed a large hematoma secondary to perforation of the deep femoral artery. The artery was ligated and the wound left open. Four days later he was admitted to the 11th Evacuation Hospital. His vital signs were normal. A routine film of the thigh revealed gas throughout the fascial planes. Exploration demonstrated a massive wine-colored edema. Counter drainage was performed and the wound was opened wide. On a continued course of penicillin and streptomycin, convalescence was uneventful. The patient probably had a clostridial wound infection rather than a true myositis. Wound cultures revealed *Cl. perfringens*.

References

1. Lindberg, R. B.; Wetzler, T.; Newton, A.; Strawitz, J. G.; and Howard, J. M.: The Early Flora of Battle Wounds in the Korean War. Preliminary Report to the Army Medical Service Graduate School, Washington, D. C., March 1954.
2. Strawitz, J. G.; Vickery, A. L.; Lindberg, R. B.; Wetzler, T.; Marshall, J. D.; Artz, C. P.; and Howard, J. M.: The Healing of Battle Wounds: Bacteriological and Histological Studies. Preliminary Report to the Army Medical Service Graduate School, Washington, D. C., July 1953.
3. Pettit, R. T.: Infections of Wounds of War. *J. A. M. A.* 73: 494, 1919.
4. Coupal, J. F.: Pathology of Gas Gangrene Following War Wounds. The Medical Department of the United States Army in the World War, Vol. XII, Sec. II. Washington, D. C., 1929.
5. North, J. P.: Clostridial Wound Infections and Gas Gangrene. *Surgery* 21: 364, 1947.
6. MacLennan, J. D.: Anaerobic Infections of War Wounds in the Middle East. *Lancet* 245: 63, 94, 123-125, 1943.



Part Three

Special Laboratory Surveys of Clinical Material

Chapter 16

Sensitivities of Ten Species of Clostridia to Penicillin, Aureomycin, Terramycin, and Chloramphenicol:

A Study of Battle Wounds in Korea*

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Battle casualties in the Korean conflict suffered less from wound infections than casualties in any previous war. During the last half of the war, gas gangrene was a rarity; death from gas gangrene was almost unknown.¹ Success in preventing severe, clostridial infections was probably due to the repair of arterial injuries, early and adequate débridement of wounds, and early and consistent use of antibiotics. It was demonstrated in Korea,² as had been shown in earlier wars,^{3,4} that clostridia were present in approximately 46 per cent of wounds at the time of primary débridement.

The average time-lag from injury to arrival at the 46th Surgical Hospital (Mobile Army) was only 3.5 hours on the eastern front during the last 18 months of the war; the subsequent time-lag before reaching the operating room being only 1.0 to 1.5 hours.

Thus, in the latter half of the war, short evacuation times and small casualty loads permitted optimal surgical care and resulted in a low incidence of severe clostridial infections.

In earlier wars, mass casualties such as occurred at times in Korea, or which might occur with atomic warfare and civilian disasters, would not permit the early or individualized attention given to the soldiers during the period of this study. For these reasons, as well as for the improvement of the care of soldiers in Korea, a re-evaluation of the antibiotic program in the combat zone was undertaken. Throughout the latter part of the war, penicillin (300,000 to 600,000 units) was administered intramuscularly as the casualty reached a battalion aid station. Following subsequent surgery, casualties

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routinely received penicillin (300,000 units) every 12 hours. Those casualties having intraperitoneal injuries, arterial injuries, or intrathoracic injuries, also received 0.5 gm. of streptomycin every 12 hours. Aureomycin was available, but it was not used very often.

Material and Methods

In a previous study, 11 wounds inflicted during the Korean conflict were examined bacteriologically throughout the first week of healing.⁵ This was done by culture of tissue excised from the open wound at the time of primary débridement and every 2 days thereafter during the ensuing week. Cultures obtained included 56 clostridial isolates and numerous aerobic organisms.

In this study, the sensitivities of the clostridial isolates to penicillin, Aureomycin, Terramycin, and chloramphenicol are presented and correlated with human serum levels of these antibiotics.⁶⁻⁸

Isolates were grown for 18 hours at 37° C. in tubes of thioglycollate broth. One ml. of this culture was transferred to a flask containing 100 ml. of thioglycollate broth. This mixture served as seed broth. The antibiotics were prepared in thioglycollate broth in such a manner that the mixture of 2 ml. of the antibiotic solutions and 2 ml. of the seed broth gave the following concentrations of antibiotics in units/ml or μ gms/ml.

Penicillin: .025, .05, 0.1, 0.2, 0.4, 0.8, 1.6, 3.2, 6.4 units/ml.

Aureomycin: .0125, 0.25, .05, 0.1, 0.4, 0.8, 1.2, 1.8, 3.2 μ gm/ml.

Terramycin: .025, .05, 0.1, 0.2, 0.4, 0.8, 1.2, 1.6, 3.2, 6.4 μ gm/ml.

Chloramphenicol: 0.5, 1, 2, 4, 6, 8, 12, 16, 20, 32 μ gm/ml.

The tubes were incubated at 37° C. for 20 hours. At this time, the tube of lowest concentration showing no visible growth was read as containing the concentration of antibiotic which caused complete inhibitions of growth. A control tube containing 2 ml. of seed broth and 2 ml. of thioglycollate broth was used for each organism tested; and it was incubated for the same length of time and at the same temperature.

Table 1 shows that sensitivities of the 56 clostridial isolates to the antibiotics, penicillin, Aureomycin, Terramycin, and chloramphenicol, follow normal distribution curves. The range of inhibition for penicillin is 0.2 to 6.4 units/ml; Aureomycin, 0.0125 to 0.8 μ gm/ml; Terramycin, 0.55 to 3.2 μ gm/ml; and chloramphenicol, 1.0 to 32.0 μ gm/ml. Those dilutions of the antibiotic which did not inhibit were omitted.

Figure 1 shows human serum concentrations following the administration of from 300,000 to 600,000 units of procaine penicillin G, and correlates these levels with the number of clostridial isolates inhibited by increasing concentrations of penicillin.⁶ A comparison of serum

Table 1. Distribution of Sensitivities of Clostridial Isolates to Four Antibiotics

Clostridium Species	Total Num-ber	Penicillin (units/ml)						Aureomycin (μgm/ml)						Terramycin (μgm/ml)						Chloramphenicol (μgm/ml)												
		.2	.4	.8	1	6	3	2	6	.4	.0125	.025	.05	.1	.2	.8	.05	.1	.2	.4	.8	3	2	1	2	4	6	8	12	16	20	32
Cl. bifermentans	17		2	2	2	10	3			1	2	12	2			1	11	3	2				2	4	5	3				3		
Cl. butyricum	1						1					1					1						1		1							
Cl. lentoputrescens	1	1								1																						
Cl. novyi	8		2	2	2	1	1	2			2	3	3			1	1	4	2	1		1			4	1	1	1		1		
Cl. paraputrificum	2					2						1	3	2			1	1	3	1				1	2	2				2		
Cl. perfringens	7	1	2	2	2	2				1	1	1	1			1	1	1						1	1	2	2					
Cl. sordelli	2				1	1																					1	1		3		
Cl. sporogenes	11	1	3	2	2	1	2				3	7	1	1		1	6	2	1	1				1	3	1	3	2	2	1	1	
Cl. tetanomorphum	2		1	1								1	1			1	1								1	1	1					
Cl. unclassified	5		3			1	1				4			1		1	3	1						1	2	1				1		
Total	56	3	13	10	19	6	5	1	4	25	18	7	1	7	24	12	7	5	1	3	5	15	14	3	4	7	3	2				

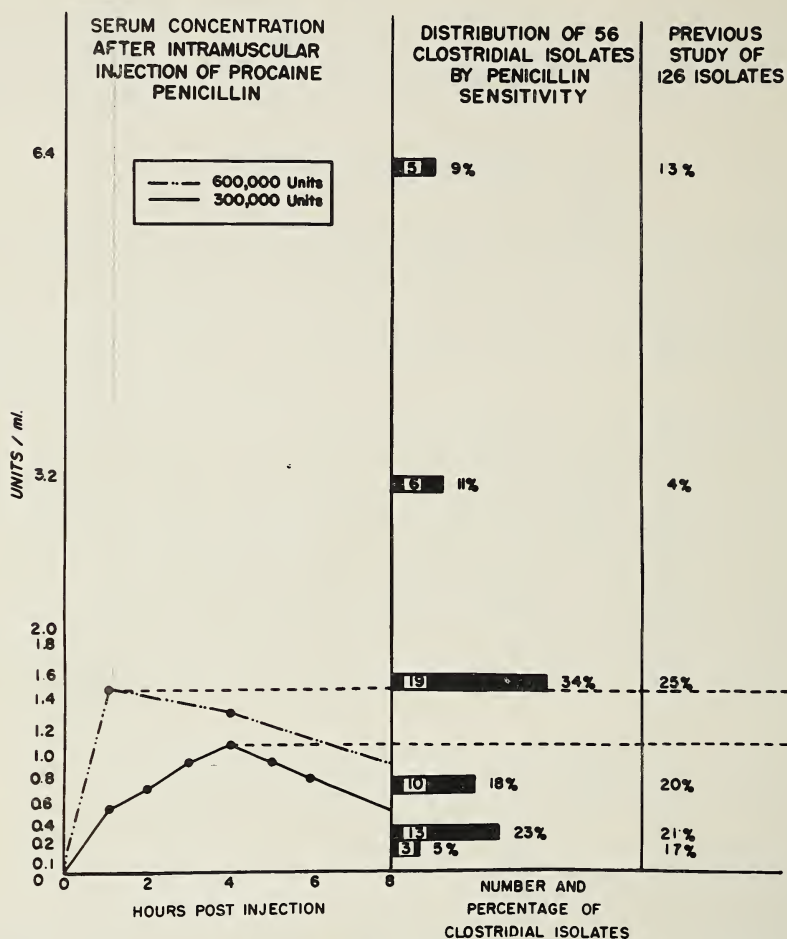


FIGURE 1. Sensitivity of the Clostridial Isolates to Penicillin. The percentages in the column on the far right are the results of an earlier study of battle wounds at the time of primary débridement.² The dotted line represents the maximal concentrations of the drug.

levels in Figure 1 with concentrations of penicillin necessary for inhibition demonstrates that approximately 80 per cent of the clostridial isolates are inhibited by the highest serum level reached after the administration of 600,000 units. Since this level is maintained for a short period only, the percentage of organisms falling outside of the range of inhibition would probably become greater when the entire absorption curve is considered.

Following the administration of 300,000 units of procaine penicillin, 46 per cent of the organisms (as indicated by the *in vitro* assay) would be inhibited by the maximal concentration found in the serum. The sensitivities in this study correspond closely with the results of

Sensitivities of Ten Species of Clostridia

Table 2. Inhibition of Clostridial Isolates by Various Antibiotics

Drug	Dosage	Percentage of Clostridial Isolates Inhibited by Maximal (Serum) Concentration	
		56 Isolates of This Study	126 Isolates of Previous Study
Procaine penicillin	300,000 units	46%	58%
Procaine penicillin	600,000 units	80%	83%
Aureomycin	0.5 gram	100%	97%
Terramycin	0.5 gram	98%	84%
Chloramphenicol	0.5 gram	66%	66%
Chloramphenicol	1.0 gram	78%	87%

a previous study of 126 clostridial isolates from many battle wounds at the time of the initial débridement.² According to the previous study, 83 per cent and 58 per cent of the clostridia would be inhibited by the maximal serum concentration following the two dosages (Table 2).

Following the oral administration of 0.5 gm. of Aureomycin, the serum concentration ranged between 1.5 and 1.7 $\mu\text{gm/ml}$ for 6 hours (Fig. 2).⁷ This concentration proved inhibitory, *in vitro*, to 100 per cent of the 56 clostridial isolates in this study and to 97 per cent of the 126 isolates in an earlier study.

Following the oral administration of 0.5 gm. of Terramycin, the serum concentration ranged around 1.2 $\mu\text{gm/ml}$ between 1 and 4 hours after ingestion (Fig. 3).⁷ As indicated by the *in vitro* studies, this concentration was inhibitory to 98 per cent of the 56 clostridial isolates in the study, and to 84 per cent of the 176 clostridial isolates studied previously.

Following the oral administration of 0.5 gm. of chloramphenicol, the serum concentration rises to 6 $\mu\text{mg/ml}$ after 3 hours. The concentration then gradually decreases (Fig. 4).⁸ This maximal concentration, *in vitro*, inhibited the growth of 66 per cent of the 56 isolates in this study; this finding confirmed the earlier study. After the administration of 1.0 gm. of chloramphenicol, the serum concentration rises to 12 $\mu\text{gm/ml}$, a concentration sufficient to inhibit 78 per cent of the organisms in this study and 87 per cent in the earlier studies.

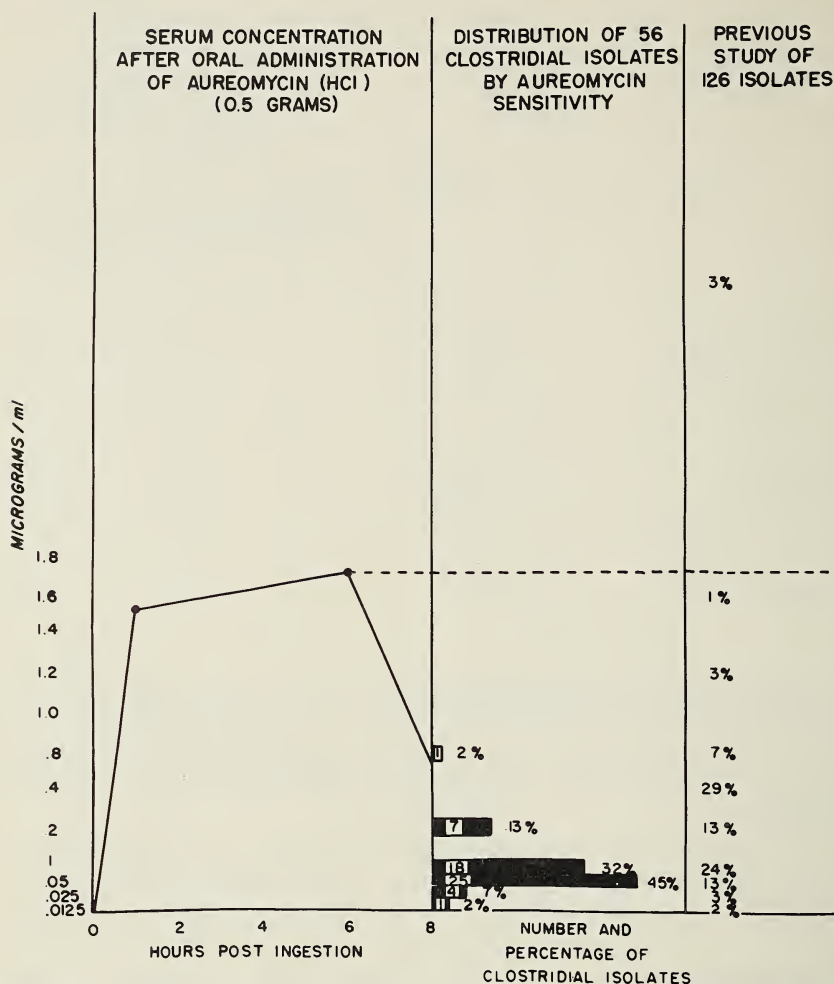


FIGURE 2. Sensitivity of the Clostridial Isolates to Aureomycin. This figure, constructed as was Figure 1, shows that in the current study all 56 clostridial isolates were inhibited by the therapeutic concentration of aureomycin.

Discussion

Antibiotic therapy has not supplanted and probably never will supplant débridement in the fundamental role of preventing or treating infections of war wounds. As an adjunct to débridement, all four of the antibiotics included in this study appear to be beneficial in preventing or minimizing clostridial infections.⁹⁻¹⁵

Sensitivities of Ten Species of Clostridia

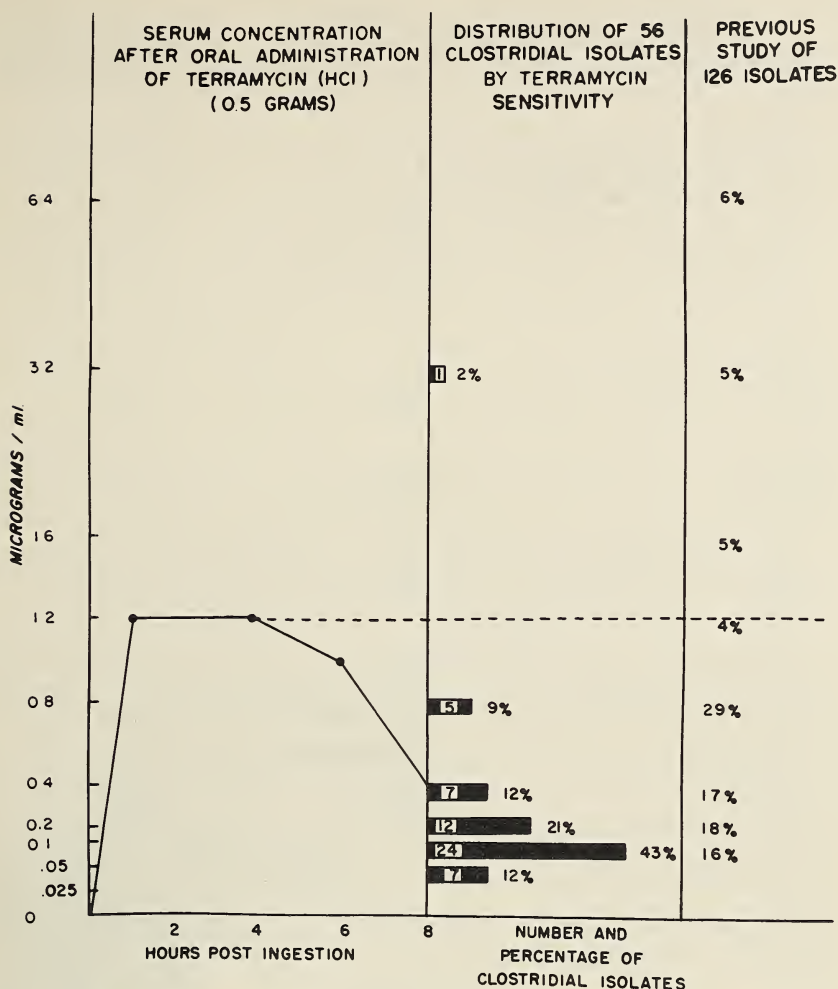


FIGURE 3. Sensitivity of the Clostridial Isolates to Terramycin.

Within the limitations of the *in vitro* sensitivity method, Aureomycin or Terramycin (of the drugs and dosages studied) would appear the drugs of choice in preventing or treating clostridial wound infections.

Experimental studies conducted in this laboratory have shown that concentrations of most antibiotics in the muscle are less than concentrations found in the serum.¹⁶ If a time should come again when—as a compromise—antibiotic therapy must be used as a bulwark to support delayed surgery of mass casualties, increased dosages of these drugs should be recommended.

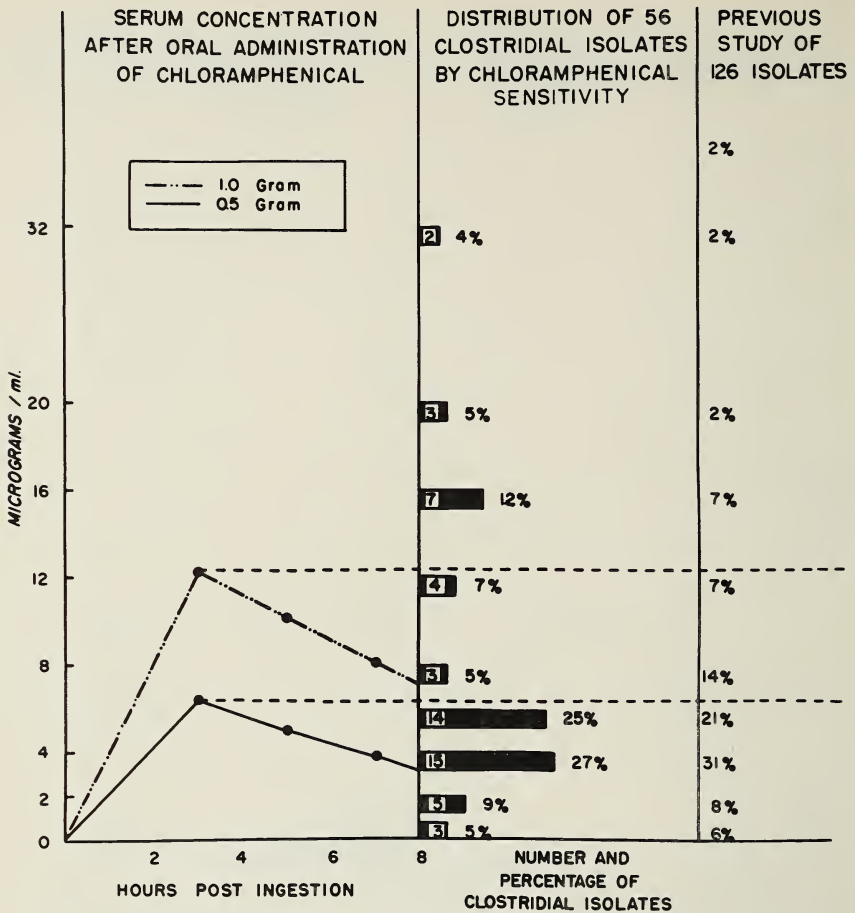


FIGURE 4. Sensitivity of the Clostridial Isolates to Chloramphenicol (Chloromycetin).

Conclusions

In vitro sensitivity studies of the flora of battle wounds in Korea suggest that Aureomycin and Terramycin are more effective than penicillin and chloramphenicol in inhibiting the growth of clostridia.

References

1. Howard, J. M., and Inui, F. K.: Clostridial Myositis—Gas Gangrene: Observations of Battle Casualties in Korea. *Surgery*, 36: 1115, 1954. (Chapter 15 of this volume.)
2. Lindberg, R. B.; Wetzler, T. F.; Marshall, J. D.; Newton, A.; Strawitz, J. G.; and Howard, J. M.: The Bacterial Flora of Battle Wounds at the Time of Primary Débridement: A Study of the Korean Battle Casualty. *Ann. Surg.* 141: 369, 1955. (Chapter 18 of this volume.)
3. Stoddard, J. L.: The Occurrence of Significance of *B. welchii* in Certain Wounds. *J. A. M. A.* 71: 1400, 1918.
4. MacLennan, J. D.: Anaerobic Infections of War Wounds in the Middle East. *Lancet*, 245: 123, 1943.
5. Strawitz, J. G.; Wetzler, T. F.; Marshall, J. D.; Lindberg, R. B.; Howard, J. M.; and Artz, C. P.: The Bacterial Flora of Healing Wounds: A Study of the Korean Battle Casualty. *Surgery*, 37: 400, 1955. (Chapter 19 of this volume.)
6. Romansky, Monroe J., George Washington University School of Medicine, Washington, D. C. Personal communication.
7. Welch, H.; Hendricks, F.; Price, C.; Randall, W.: Comparative Studies on Terramycin and Aureomycin. *J. Am. Pharm. Assn.* 39: 185, 1950.
8. Burnell, J. M., and Kirby, W. M. H.: Serum Concentration on Chloramphenicol Following Intravenous and Intramuscular Administration. *J. Lab. & Clin. Med.* 38: 234, 1951.
9. Smith, L. D.: Clostridia in Gas Gangrene. *Bact. Rev.* 13: 233, 1949.
10. Ulio, J. A., and Marshall, G. C.: 1945 Notes on Care of Battle Casualties. War Department Technical Bulletin, T. B. Med. 147, War Medicine, pp. 234-248.
11. Hac, L. R.: Experimental *C. welchii* Infection. IV. Penicillin Therapy. *J. Infec. Dis.* 74: 164, 1944.
12. Taylor, W. I., and Novak, M. V.: Prophylaxis of Experimental Gas Gangrene in Mice. *J. Bact.* 61: 571, 1951.
13. Altemeier, W. A.; Furste, W. L.; and Culbertson, W. R.: Chemotherapy in Gas Gangrene. *Arch. Surg.* 55: 668, 1947.
14. Sandusky, W. R.; Keeble, C. F.; Wharton, W. P.; and Taylor, R. N.: An Evaluation of Aureomycin and Chloromycetin in Experimental *Clostridium welchii* Infection. *Surgery*, 28: 632, 1950.
15. Altemeier, W. A.; McMurrin, J. A.; and Alt, L. F.: Chloromycetin and Aureomycin in Experimental Gas Gangrene. *Surgery*, 28: 621, 1950.
16. Marshall, J. D., and Strawitz, J. G.: A Comparison of Antibiotic Levels in Serum and Muscle of Rabbits. (To be published.)

Chapter 17

The Early Bacterial Flora of Wounds in the Korean Conflict

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Introduction

A study of the bacterial contamination of battle wounds, as it occurred during the first 4 to 8 hours after injury, was made at the 46th Surgical Hospital on the east-central front in Korea during the last year of the Korean conflict. Cases were observed during August and September 1952, and from December through February 1953. Various reports in the literature regarding World War I and World War II, for the most part, included cases in which less recent wounds were considered.¹⁻³ Under these circumstances, infection was far more likely to be well established at the time of culture than it was in the case of wounds observed in this study. The earlier reports showed that *Clostridia* were present in from 60 to 80 per cent of the wounds within 1 to 4 days after injury. Most of these observations were made on soldiers who had not received intensive and early penicillin prophylaxis and therapy to the extent that it was employed in the Korean conflict.

The incidence of gas gangrene was lower in World War II than in World War I. In both conflicts, such infections were extremely rare in comparison with the extensive contamination of wounds by pathogenic and saprophytic *Clostridia*. In the Korean conflict, the exact incidence rate of gas gangrene and of anaerobic cellulitis was not ascertained, but it was evidently lower than in the previous wars. This improvement has been ascribed largely to the rapid evacuation of the wounded, prompt and thorough surgical débridement coupled with more effective shock therapy, and the extensive use of broad-spectrum antibiotics. It is important, however, that the degree and nature of bacterial contamination of these wounds be determined as a part of the basis for evaluating the role of wound infections in modern war surgery and for maintaining a perspective on the survival potentialities of

patients having Clostridia and receiving antibiotic prophylaxis soon after wounding. This report presents an analysis of the bacterial flora of wounds incurred by troops during the latter part of the Korean conflict and some observations on flora of the environment of these troops.

Materials and Methods

Sources

Of the two series of cases observed during 1952 and 1953, Series A was composed of 39 wounded patients who were studied during August and September 1952, and Series B consisted of cultures taken on 87 cases from November 1952 through February 1953. Specimens from these patients were cultured at the time of arrival and débridement at the surgical hospital. The specimens represented mainly wound cultures taken 4 to 6 hours after injury, with follow-up cultures in some instances. The cases thus illustrate wound contamination and early proliferation of bacteria, rather than infection. Principal emphasis was placed on isolation and identification of Clostridia since these organisms, in addition to their classical role in gas gangrene, offer a potential source of toxins which may contribute to the development of secondary shock and to the kidney damage connected with posttraumatic anuria.

The speed of routine evacuation of patients limited most observations to initial débridement specimens in which typical infection had not yet appeared. Cases selected included principally arm and leg wounds.

In addition to tissues, the flora of the skin of patients and troops, of clothing worn in combat, and of the soil of the combat zone were studied to determine the relations of such organisms to the flora of contaminated wounds. Blood cultures were obtained in some cases of both series at the time of admission to the hospital. In Series A, 30 men arrived at the hospital in time to permit cultures to be obtained at an average interval of 4.5 hours following injury. However, 13 per cent of the patients arrived 6 or more hours following injury. In three patients, cultures were taken 1 or more days after wounding.

Technics of Culture

Blood cultures were obtained using evacuated rubber-stoppered bottles with reduced oxygen and increased CO₂ tension over a trypticase soy broth. Skin cultures were taken by means of swabs moistened in broth, rubbed over the lateral aspect of the lower leg, and inoculated in chopped heart broth. Clothing samples were placed in dry, sterile vials. Wounds were sampled during débridement by collecting portions of tissue on sterile gauze pads as they were removed by the surgeon.

These samples were planted at once in a modified Robertson's cooked meat medium in screw-capped tubes. This medium was found to maintain virtually all anaerobic and aerobic flora when held at room temperature for at least 3 months. The inoculated meat-broth tubes were incubated for 48 hours at 37° C. They were then transferred to blood-agar plates containing yeast extract. Inhibitors were added to media to suppress interfering aerobic flora. These included sodium azide (0.2 per cent), chloral hydrate (0.2 per cent), and at times sorbic acid (0.15 per cent). Subcultures to thioglycollate broth containing sorbic acid and polymyxin B were made when necessary, with repeated platings to isolate pure cultures of anaerobes. Anaerobiasis was maintained in Brewer jars, with hydrogen introduced at 200 mm. pressure. Differentiating reactions with pure cultures were carried out in thioglycollate broth or semisolid medium.

Particular emphasis was placed on isolation of *Cl. perfringens*. Presumptive recognition of this species was made with the acrolein reaction.⁴

Classification of Clostridia

Clostridia were classified when possible according to the modified Spray classification as presented in *Bergey's Manual of Determinative Bacteriology*, 6th edition.⁵ In cases where the characterization of strains in this system was incomplete or ambiguous, the classification of Reed and Orr⁶ was employed in conjunction with the Bergey outline. Four principal categories of strains were differentiated as shown in Table 1. Aerobic flora were classified chiefly according to the scheme of *Bergey's Manual*.

Table 1. Categories of Clostridia

Group 1	stormy fermentation in iron-milk	<i>Cl. perfringens</i> ,* <i>butyricum</i> , <i>beijerinckii</i> , <i>pasteurianum</i> , <i>multifermentans</i> , and <i>aerofaecium</i> **
Group 2	acid, coagulation and gas in iron-milk	<i>Cl. tertium</i> , <i>fallax</i> ,* <i>paraputrificum</i> , <i>carinis</i> ,* <i>chauvoei</i> (<i>feseri</i> *), <i>septicum</i> ,* and <i>sphenoides</i>
Group 3	iron-milk digested	<i>Cl. novyi</i> ,* <i>sporogenes</i> , <i>sordelli</i> ,* <i>bifermentans</i> , <i>histolyticum</i> ,* <i>lentoputrescens</i> , <i>parabotulinum</i> ,* and <i>putrefaciens</i>
Group 4	no specific action in iron-milk	<i>Cl. tetani</i> ,* <i>tetanomorphum</i> , <i>cochlearium</i> , <i>difficile</i> ,* <i>capitovale</i> , <i>viscificiens</i> , and <i>filiforme</i>

*Organism pathogenic for guinea pigs; pathogenicity of strains may vary.

**Some strains pathogenic for guinea pigs.

Results of Observations, Series A, Summer 1952

The two periods of observation, Series A and Series B, showed culture results which varied so markedly that it was desirable to present them separately. The major distinction between the two was a marked decrease in the relative proportion of *Clostridia* isolated in the winter in comparison with the number found in wounds during the summer.

Of the 39 patients studied in Series A, 33 had one or more tissue specimens collected at débridement. Six patients had blood cultures taken simultaneously; while six other patients had only blood cultures. In attempts to ascertain sources of contamination, 29 soil samples were collected from the combat zone, from hillsides and rice paddies. The causative agents of wounds, when known, were tabulated in Table 2. Included in this series were 21 cases of leg wounds of varying severity, 11 arm and shoulder injuries, and 7 head and neck or body injuries.

Anaerobic Flora of Tissue and Embedded Fragments at Débridement, Series A

Cultures were made of 94 tissue specimens from the 33 patients. Twenty-eight patients harbored *Clostridia* in tissues, with a total of 63 tissue samples which were positive. Five samples of cloth were removed from various wounds during débridement and *Clostridia* were isolated, although the tissues in one of these wounds did not reveal *Clostridia*. A shell fragment removed and cultured showed two species of *Clostridia*; and the same species were also isolated from adjacent tissues. Among these 33 patients, 84.8 per cent showed positive cultures of *Clostridia* from one or more débrided tissues. There were 125 strains recovered, including 19 species. *Clostridia* were isolated in 67 per cent of the tissue specimens. An average of 4 *Clostridial* species per patient was present in cases harboring those organisms; while 2 strains were isolated per tissue sample. The five pieces of embedded clothing showed 1.8 strains per sample, representing 9 strains and 5 species. One shell fragment from deep muscle was cultured and showed two species of *Clostridia*.

Clostridia Isolated in Blood Cultures, Series A

Collection of 15 blood cultures from 12 patients showed *Clostridia* in 11 patients. Strains of *Clostridia* isolated totaled 19, including four species and four aerobic organisms.

The bacterial flora of the Korean soil in the combat area was studied in both the summer and the winter series. No essential difference was noted in the flora of the soil in these two seasons.

The anaerobic flora of tissues and blood cultures in Series A is summarized in terms of total strains isolated (Table 3).

Table 2. Causes of Wounds and Clostridia Present in 32 Casualties in Korea, Series A, August–September 1952

Cause of Wound	Number of Patients	Patients with Clostridia	Average Number of Species per Patient	Specimen		Total Strains Recovered	Number of Species	Average Species per Positive Specimen
				Number	Clostridia Present			
Small arms	5	5	1.8	11	6	10	5	1.7
Artillery	6	5	3.0	18	16	29	8	1.7
Mortar and grenade	15	14	4.8	47	31	67	17	2.16
Land mine	6	5	6.0	26	17	30	10	1.8
Total	32	29		102	70	136		

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Table 3. Anaerobic Flora of Wounds and Blood, Series A, August–September 1952

Clostridium Species	Source				Total Number
	Tissue*		Blood		
	Number	Strains (per cent)	Number	Strains (per cent)	
Cl. sporogenes	40	29. 0	11	58	51
Cl. perfringens	29	21. 0	-----	-----	29
Cl. bifermentans	15	10. 9	4	21	19
Cl. sordelli	4	2. 8	-----	-----	4
Cl. multifementans	11	8. 0	1	5	12
Cl. novyi	10	7. 2	3	16	13
Cl. paraputrificum	5	3. 6			5
Cl. tetanomorphum	4	2. 9			4
Cl. butyricum	3	2. 2			3
Cl. tertium	3	2. 2			3
Cl. lentoputrescens	3	2. 2			3
Cl. tetani	3	2. 2			3
Cl. aerofoetidum	2	1. 4			2
Cl. histolyticum	1	0. 7			1
Cl. carnis	1	0. 7			1
Cl. cochlearium	1	0. 7			1
Cl. putrefaciens	1	0. 7			1
Cl. sphenoides	1	0. 7			1
Cl. capitovale	1	0. 7			1
Total	138		19		157

*Includes embedded clothing and fragments.

Predominant among the 19 species isolated from tissue and associated samples were *Cl. sporogenes*, *Cl. perfringens*, *Cl. bifermentans*, *Cl. multifementans*, and *Cl. novyi*. Toxigenic species (excluding *Cl. tetani*, which is not pertinent to the problem of wound infection) included *Cl. perfringens*, *Cl. novyi*, *Cl. sordelli*, and the rare *Cl. histolyticum*. The diverse Clostridial flora of these wounds was comparable to that reported in earlier wound studies.⁷ It is unusual to observe this number of cultures with all strains identifiable.

The relatively large number of patients from whom Clostridia were isolated from blood cultures was a complete surprise. Careful check on handling methods failed to reveal any obvious contaminating error in technic; and it is quite improbable that such exotic Clostridial flora should be introduced as contaminants. Blood cultures on 41 normal individuals, taken later as controls under identical circumstances as those in patients, showed no positive cultures. The predominance of *Cl. sporogenes* and the presence of *Cl. novyi*, as well as failure to isolate *Cl. perfringens*—the most common soil contaminant, would indicate that these results are valid as representing

a widespread, early bacteremia among wounded soldiers. All patients who showed positive blood cultures were seriously wounded and in shock upon arrival at the surgical hospital. In two patients, blood cultures taken 2 or more days after wounding showed *Clostridia*. Five traumatic amputations and several severe open, comminuted fractures were included in this list of patients. Other observers have reported *Clostridial* bacteremia in septic abortion,⁸ and in wound cases.⁹

Distribution of *Clostridia* by Cases

The population of *Clostridia* when correlated with the individual cases in which strains were found gave a more significant picture of the species incidence. Out of 28 cases showing *Clostridia* in tissues, the number of cases harboring each species and the total number of positive tissues are listed in Table 4.

Table 4. Incidence of *Clostridia* in Tissues of 28 Cases with Recent Wounds

<i>Clostridium</i> Species	Present in Number of Cases	Per Cent Positive for Species	Tissues Examined in Positive Cases	Number of Positive Tissues	Per Cent of Total Positive Cases
<i>Cl. sporogenes</i>	18	64.2	78	40	51.2
<i>Cl. perfringens</i>	13	46.4	58	29	50.0
<i>Cl. bifermentans</i>	10	35.7	57	16	28.0
<i>Cl. novyi</i>	7	25.0	24	10	41.6
<i>Cl. multifermentans</i>	7	25.0	37	11	29.7
<i>Cl. paraputrificum</i>	5	17.8	16	5	31.2
<i>Cl. lentoputrescens</i>	3	10.7	8	3	37.5
<i>Cl. sordelli</i>	3	10.7	9	4	44.4
<i>Cl. tertium</i>	3	10.7	6	3	50.0
<i>Cl. tetanomorphum</i>	3	10.7	15	4	26.6
<i>Cl. butyricum</i>	2	7.1	7	3	42.8
<i>Cl. tetani</i>	2	7.1	14	3	21.4
<i>Cl. aerofaecidum</i>	2	7.1	5	2	40.0
<i>Cl. carnis</i>	1	3.9	2	1	50.0
<i>Cl. capilovale</i>	1	3.9	4	1	25.0
<i>Cl. cochlearium</i>	1	3.9	3	1	33.0
<i>Cl. putrefaciens</i>	1	3.9	3	1	33.0
<i>Cl. sphenoides</i>	1	3.9	4	1	25.0
<i>Cl. histolyticum</i>	1	3.9	2	1	50.0

In numerical order, *Cl. perfringens* (46.4 per cent of the cases), *Cl. novyi* (25 per cent), and *Cl. sordelli* (10.7 per cent) were the major pathogenic species observed. The most commonly encountered *Clostridia* were *Cl. sporogenes* (64.2 per cent of the cases). Proteolytic species were preponderant among the isolates; there were 12 species which could be so classified. Among the nonpathogenic species, *Cl.*

The Early Bacterial Flora of Battle Wounds

sporogenes, *Cl. bifermentans*, *Cl. multifermentans*, and *Cl. parapatrificum* were of chief numerical importance. The proportion of tissues cultured which were positive for each species was relatively high. If positive cultures were obtained, they showed up to 50 per cent of tissue samples for a given species.

Aerobic Flora of Wounds, Series A

A limited study of the aerobic flora of Series A is summarized in Table 5, listing the species totals isolated from 90 tissue samples. The cultures, from the cooked meat broth used also for anaerobic enrichment, showed as high a proportion of aerobes as did direct aerobic procedures. The predominance of gram-negative bacilli and the infrequency of hemolytic streptococci was in marked contrast to the flora of older wounds in this conflict.

Table 5. Aerobic Flora of Recent Wounds in Korea, Series A

Microorganism*	Strains	Per Cent of Total
Streptococci, alpha hemolytic	10	5.0
Streptococci, beta hemolytic	1	.5
Streptococci, nonhemolytic	35	17.5
Staphylococci, H+ M+	3	1.5
Staphylococci, H+ M-	15	7.5
Staphylococci, H-	11	5.5
<i>Ps. aeruginosa</i>	8	4.0
<i>Proteus</i>	27	13.5
<i>Escherichia</i>	19	9.5
<i>Aerobacter</i>	27	13.5
Paracolon	10	5.0
<i>Alcaligenes</i>	3	1.5
Bacillus	29	14.5
<i>Corynebacterium</i>	2	1.0
Total	200	100.0

*H+, hemolytic

M+, mannite-fermenting

H-, nonhemolytic

M-, mannite-nonfermenting

Cases in which positive Clostridia cultures were obtained from tissues showed from one to nine species. The variation in number of Clostridia per patient in 27 instances is shown in Table 6.

Table 6. Total Clostridia in Tissues in 27 Cases, Series A

Number of Cases	Types of Species	Number of Cases	Types of Species
8	1	1	5
5	2	2	6
4	3	2	7
4	4	1	9

Series B, Winter 1952-1953

The high incidence of Clostridia in battle wounds cultured shortly after injury, and the fact that such a limited series was sampled, prompted a more exhaustive study which was initiated in December 1952, and completed in February 1953. Eighty-seven cases were examined. Tissues were cultured in 75 of these cases, while blood cultures only were taken on the remaining 12. In marked contrast to the 93.9 per cent of patients harboring Clostridia in wounds or in the blood soon after injury (Series A), only 43 of this series (57.3 per cent) showed cultures positive for Clostridia. Aerobic flora remained ubiquitous, and all cases had contaminated wounds. The reduction in recovery rate in this series does not seem to have a rational basis, although several hypotheses can be offered. In an effort to correlate the wound flora with a probable source, cultures were taken of the soil of the combat zone, clothing of troops, and the skin surfaces of the soldiers tested.

Anaerobic Flora of Tissues, Series B

A total of 443 tissue specimens, including some embedded foreign bodies, were cultured from 75 patients. Of these cases, 43 (57.3 per cent) harbored Clostridia in one or more wounds, in marked contrast to the 84.8 per cent of cases with wound cultures positive for Clostridia in Series A. Eighty-four of the tissue samples cultured showed Clostridia (19 per cent of the total). The positive series comprised 285 tissues, so that the rate of isolation of Clostridia was 29.4 per cent of these specimens. There were 127 strains isolated, including 20 identified species and a group of unidentified strains. Out of the cases positive for Clostridia, an average of 2.9 strains per patient was obtained; while among tissue samples which were positive, there were 1.4 strains per sample. Thus, in terms of patients involved and the extent and intensity of tissue contamination, the anaerobic flora of this series of cases was greatly reduced from that observed during the previous summer (Series A).

Blood Cultures, Series B

Sixty blood cultures were obtained from 53 patients. Three individuals (5.6 per cent) harbored Clostridia, in striking contrast to the 91 per cent with Clostridial bacteremia among the 12 patients in Series A. Four strains and three species were isolated.

Cause of Wound and Clostridia Present, Series B

The degree of wound contamination in relation to wounding agent was observed in this series in a manner similar to that recorded in Series A. Results are summarized in Table 7.

Table 7. Causes of Wound and Strains of Clostridia Present in 28 Patients, Series B, Winter 1952-1953

Cause of Wound	Number of Patients with Clostridia	Average Number of Species per Patient	Specimen			Total Strains Recovered	Average Species per Positive Specimen
			Total Number	Positive for Clostridia			
				Number	Per Cent		
Mortar shells	7	2.5	51	13	25.4	21	1.6
Grenades	6	2.0	36	14	38.9	16	1.1
Rifle and machine gun fire	8	2.8	40	22	55.0	39	1.7
Artillery shells	17	2.0	82	27	32.9	35	1.6
Total	38		209	76		111	

The earlier observation of a preponderance of contamination occurring with high-explosive and mortar-shell injuries, as contrasted to the effect of small-arms fire, was not borne out in this series. It is possible that the heavier clothing worn during the winter reduced the amount of contamination introduced at the time of wounding. The initial contamination of a wound is assumed to be imparted chiefly by the blast effect of the missile. No distinction associated with variation in this effect was apparent in this series. Rifle and machine-gun missiles occasioned the most severely contaminated wounds (55 per cent), while shell and grenade injuries were positive in from 25.4 to 38.9 per cent of the cases.

Total Number of Clostridial Strains Observed, Series B

The Clostridial flora, in terms of strains isolated from tissues and blood, is shown in Table 8.

The predominant species were not significantly different from those observed in the summer series. *Cl. perfringens*, *Cl. sporogenes*, and *Cl. novyi* were the predominant organisms. *Cl. bifermentans* ap-

Table 8. Anaerobic Flora of Wounds and Blood, Series B, December–February 1952–1953

<i>Clostridium</i> Species	Tissues		Blood	
	Number of Strains	Per Cent of Total	Number of Strains	Per Cent of Total
<i>Cl. sporogenes</i>	25	19.6	2	50.0
<i>Cl. perfringens</i>	23	18.1	1	25.0
<i>Cl. novyi</i>	7	5.5	-----	-----
<i>Cl. bifermentans</i>	4	3.1	-----	-----
<i>Cl. sordelli</i>	4	3.1	-----	-----
<i>Cl. multi fermentans</i>	3	2.3	-----	-----
<i>Cl. lentoputrescens</i>	6	4.7	1	25.0
<i>Cl. paraputrificum</i>	7	5.5		
<i>Cl. tertium</i>	4	3.1		
<i>Cl. tetani</i>	5	3.9		
<i>Cl. carnis</i>	5	3.9		
<i>Cl. capitovale</i>	4	3.1		
<i>Cl. tetanomorphum</i>	3	2.3		
<i>Cl. fallax</i>	2	1.5		
<i>Cl. cochlearium</i>	2	1.5		
<i>Cl. aerofoetidum</i>	2	1.5		
<i>Cl. sphenoides</i>	2	1.5		
<i>Cl. difficile</i>	1	.8		
<i>Cl. butyricum</i>	1	.8		
<i>Cl. putrefaciens</i>	2	1.5		
unclassified or unidentified	15	11.8		
Total	127		4	

peared less often, with *Cl. sordelli* being proportionately more common. In no other series did these closely related organisms occur with equal frequency. *Cl. carnis* was more common in the winter series than in the summer series. *Cl. fallax* and *Cl. difficile* were first encountered in Series B. The latter strain is extremely rare in war wounds, and it may not have been observed previously.¹⁰ A significantly large group of unidentifiable strains was observed. These strains included both toxigenic and non-toxigenic organisms, many of which were microaerophilic. Their taxonomic position could not be defined on the basis of the current classification.

The isolates from the blood, as regards Clostridia, were limited to three species and four strains. As pointed out previously, the wide discrepancy in these two series poses an unanswered question as to the actual over-all incidence of bacteremia with Clostridia in the period soon after injury.

Distribution of Clostridia in Cases, Series B

Table 9 gives a summary of the case incidence and frequency of occurrence in tissues of the various species of Clostridia in Series B. The two principal species, *Cl. perfringens* and *Cl. sporogenes*, were present in almost one-third of the cases; of the remainder, species of principal interest included *Cl. novyi*, *Cl. lentoputrescens*, *Cl. parapatrificum*, and *Cl. carnis*. Case incidence of *Cl. novyi* was significantly lower than in Series A. It was also distinctly below the level encountered in most studies of wounds in World War II in North Africa and Europe.³

The percentage of tissue specimens positive for each species was much lower than in Series A. This may reflect a decreased concentration of Clostridia in wounded tissue incurred under winter conditions.

Aerobic Flora of Wounds, Series B

A summary of the aerobes isolated from a representative set of 42 cases in Series B is shown in Table 10. The principal aerobes were streptococci (Group D), alpha hemolytic and nonhemolytic strains, staphylococci and *Bacillus* species. *Corynebacterium* strains were present in 50 per cent of the cases. The numbers of *Proteus* strains and coliform bacilli were unexpectedly low, and *Pseudomonas aeruginosa* was found in only one case. These results are significantly at variance with the findings of Series A. Cultures during the summer rarely showed hemolytic streptococci or staphylococci, but organisms of the *Enterobacteriaceae* group were more common. *Bacillus* species were relatively common in both series. None of Group A streptococci were isolated, nor were they found in the remaining 45 cases cultured.

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Table 9. Incidence of Clostridial Species in Tissues of 43 Cases Showing Cultures Positive for Clostridia, Series B

<i>Clostridium</i> Species	Present in Num- ber of Cases	Strains Recovered from All Positive Cases	Per Cent Positive for Species	Tissues Cultured in Positive Cases	Per Cent of Positive Tissues
<i>Cl. perfringens</i>	13	23	30. 2	78	29. 4
<i>Cl. sporogenes</i>	13	25	30. 2	69	36. 2
<i>Cl. novyi</i>	5	7	11. 5	26	26. 9
<i>Cl. multifementans</i>	2	3	4. 6	10	30. 0
<i>Cl. bifermentans</i>	3	4	6. 9	17	23. 5
<i>Cl. sordelli</i>	3	4	6. 9	16	25. 0
<i>Cl. lentoputrescens</i>	6	6	13. 9	31	19. 3
<i>Cl. paraputrificum</i>	7	7	16. 2	31	22. 5
<i>Cl. tetanomorphum</i>	2	3	4. 6	7	42. 8
<i>Cl. tertium</i>	4	4	6. 9	13	30. 7
<i>Cl. difficile</i>	1	1	2. 3	2	50. 0
<i>Cl. butyricum</i>	1	1	2. 3	14	7. 1
<i>Cl. putrefaciens</i>	2	2	4. 6	8	25. 0
<i>Cl. capitovale</i>	4	4	6. 9	36	11. 1
<i>Cl. fallax</i>	2	2	4. 6	10	20. 0
<i>Cl. tetani</i>	5	5	11. 5	33	15. 1
<i>Cl. cochlearium</i>	2	2	4. 6	7	28. 5
<i>Cl. aerofoetidum</i>	2	2	4. 6	8	25. 0
<i>Cl. sphenoides</i>	2	2	4. 6	6	33. 3
<i>Cl. carnis</i>	5	5	11. 5	21	23. 8
unclassified	12	15	27. 9	75	20. 0

Table 10. Incidence by Case of Aerobic Flora in 42 Cases Positive for Clostridia, Series B

Microorganism	Positive Cases	Per Cent Positive
Streptococci, beta hemolytic*	39	92. 8
Streptococci, alpha hemolytic	22	52. 3
Streptococci, nonhemolytic	37	88. 0
Straphylococci, hemolytic	29	69. 0
Straphylococci, nonhemolytic	27	64. 0
Bacillus	35	83. 3
<i>Corynebacterium</i>	21	50. 0
<i>Pseudomonas</i>	1	2. 3
<i>Proteus</i>	9	21. 4
Coliform bacilli	14	33. 3
Paracolon bacilli	6	14. 2
<i>Gaffkya tetragenes</i>	1	2. 3
<i>Alcaligenes</i>	4	9. 5
<i>Lactobacillus</i>	1	2. 3

*27 strains among this group were characterized serologically as Group D; and no Group A streptococci were isolated.

Clostridial Flora of Skin, Clothing, and Soil

The bacterial flora of intact skin constituted an obvious potential source of wound contamination. Two sets of skin swabs were cultured, one from patients upon admission, the other from members of an infantry company returning from the battle line for shower baths. During the winter study, 66 samples were collected. Results of the two sets of cultures were pooled.

Seventeen samples of clothing, including fatigues, socks, and woolen underwear, were collected from patients on admission to the hospital.

During both Series A and Series B, a total of 50 samples of soil was collected. Specimens were obtained from rice paddies, hillsides, bunkers, and a variety of areas occupied by the line companies along an active, division front. Of course, Clostridia were most numerous in the soil; and all specimens showed strains if carefully handled to control interfering growth.

The distribution of strains isolated from these three environmental sources are shown in Table 11.

The Clostridia found in the three environmental areas (skin, clothing, and soil) presented data of interest when it was compared with the flora isolated in wounds shortly after injury. In proportions of the principal species, *Cl. perfringens*, *Cl. sporogenes*, and *Cl. novyi* isolated from the skin showed a close resemblance to the bacterial flora of wounds studied simultaneously. The relatively small number of samples and the relatively low isolate rate (46.9 per cent of 66 samples which were positive) may explain the failure to obtain some species observed in wounds, including *Cl. lentoputrescens* and *Cl. carnis*. Among the clothing samples, 66 per cent were positive for Clostridia. The preponderance of *Cl. perfringens* and the absence of various species observed in wounds, would indicate that *Cl. perfringens* may not be the major source contributing Clostridia to wounds. The absence of *Cl. novyi*, *Cl. multifementans*, and *Cl. lentoputrescens* is noteworthy. When the soil was considered, the preponderance of *Cl. perfringens* was of particular interest because it was not as prominent as it was in the flora of recent wounds. *Cl. butyricum* was much more common in soil than in wounds. Although any of the three sources could contribute to the flora of the wound, the data indicated that in terms of relative incidence, the strains isolated on the skin showed a ratio comparable to the principal species occurring in wounds. Therefore, strains found on the skin are always suspected as a source of contamination.

The total of 25 species recognized among these was greater than the total number of species found in any tissue series; and several species were not isolated from tissues, including *Cl. subterminale*, *Cl. pasteurianum*, and *Cl. beijerinckii*.

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Table 11. Clostridial Strains Isolated from Skin, Clothing, and Soil in Korea, 1952-1953

<i>Clostridium</i> Species	Skin		Clothing		Soil	
	Total Number	Per Cent of Total Strains	Total Number	Per Cent of Total Strains	Total Number	Per Cent of Total Strains
<i>Cl. perfringens</i>	10	20.8	9	32.1	44	32.1
<i>Cl. sporogenes</i>	11	22.9	3	10.6	24	17.5
<i>Cl. novyi</i>	3	6.2	0	-----	4	2.9
<i>Cl. multi fermentans</i>	3	6.2	0	-----	15	10.9
<i>Cl. bifermentans</i>	1	2.0	2	6.9	15	10.9
<i>Cl. sordelli</i>	0	-----	1	3.5	1	.7
<i>Cl. lentoputrescens</i>	0	-----	0	-----	3	2.1
<i>Cl. paraputrificum</i>	2	4.1	0	-----	1	.7
<i>Cl. tetanomorphum</i>	1	2.0	1	3.5	3	2.1
<i>Cl. tertium</i>	1	2.0	0	-----	2	1.4
<i>Cl. difficile</i>	0	-----	0	-----	1	.7
<i>Cl. butyricum</i>	0	-----	3	10.6	9	6.5
<i>Cl. putrefaciens</i>	1	2.0	0	-----	0	-----
<i>Cl. capitovale</i>	1	2.0	0	-----	0	-----
<i>Cl. fallax</i>	0	-----	1	3.5	0	-----
<i>Cl. tetani</i>	1	2.0	1	3.5	0	-----
<i>Cl. cochlearium</i>	1	2.0	1	3.5	1	.7
<i>Cl. aerofaecidum</i>	2	4.1	0	-----	4	2.9
<i>Cl. carnis</i>	0	-----	0	-----	2	1.4
<i>Cl. sphenoides</i>	0	-----	0	-----	1	.7
<i>Cl. histolyticum</i>	0	-----	1	3.5	0	-----
<i>Cl. parabotulinum</i>	1	2.0	1	3.5	0	-----
<i>Cl. subterminale</i>	1	2.0	0	-----	0	-----
<i>Cl. pasteurianum</i>	1	2.0	0	-----	2	1.4
<i>Cl. beijerinckii</i>	2	4.1	0	-----	0	-----
unclassified	5	10.4	4	14.2	5	3.6
Total	48		28		137	

Skin cultures of individuals numbered 66; skin cultures positive for Clostridia, 31.
 Samples of clothing cultured, 17; samples positive for Clostridia, 13.
 Samples of soil cultured, 50; samples positive for Clostridia, 50.

Discussion

Studies reported were unusual in observations on war wounds, particularly in their emphasis on cultures obtained at from 4 to 8 hours after injury and in the fact that most cases received from 300,000 to 600,000 units of penicillin intramuscularly shortly after injury. Almost all wounds were contaminated; but the incidence of Clostridia varied widely between wounds in Series A and in Series B. Of the 33 cases in Series A in which tissues were cultured, 84.8 per cent har-

bored Clostridia, in contrast to 57.3 per cent of the 75 cases in Series B. Of the cases in Series B diagnosed as Clostridial infection, only 29.4 per cent of the biopsied tissues showed Clostridia. This was in contrast to 67 per cent of all tissues which were positive in the summer series. A group of blood cultures in the summer series was positive for Clostridia in 91 per cent of the cases, while only 5 per cent of the winter series showed Clostridia. This striking discrepancy has no valid explanation at present.

Blood cultures were more apt to be positive in the case of severe wounds with shock; and there was additional indication that the patients who failed to receive penicillin were more prone to bacterial invasion of the blood. Reports included little data on Clostridial bacteremia; and the proportion of cultures which was positive appeared to be unusually high.

The nature of the wounding agent was associated with marked differences in the degree of wound contamination in Series A, shell fragments being far more prone to produce a contaminated wound than rifle fire. In Series B, however, there was little difference in degree of contamination in wounds due to grenades, mines, rifle or artillery fire.

The time lapse between wounding and débridement was much shorter during the last year of the Korean conflict than it was in previous wars. Apparently the mean time of 4.5 hours in the summer and 7.9 hours in the winter was within the period in which bacterial proliferation is present, but not sufficiently long to initiate the tissue defense against infection. Wounds were apt to be far more heavily contaminated when cultured one day after injury.

The primary emphasis in these observations was placed on the anaerobic flora. The aerobic flora was characteristic in Series A, showing a predominance of *Enterobacteriaceae*, *Bacillus* species, and nonhemolytic streptococci. In Series B, however, there were more staphylococci and far more hemolytic streptococci, chiefly Group D. By contrast, the various *Enterobacteriaceae* were less common than in Series A. *Bacillus* strains were prominent in both series. The failure to isolate even one strain of Group A streptococci was significant.

The anerobic streptococci isolated were numerous enough to be of interest, but could not be studied effectively. They were grouped in the table with streptococci. Studies should be made of these organisms at some future time.

When environment was considered, the skin of patients showed a flora quantitatively similar to that of the wounds. Among anaerobes, *Cl. perfringens* was predominant in the samples from soil and clothing. *Cl. multifementans* and *Cl. bifermentans* were more common

in the soil than in wounds. The latter two organisms, however, were a conspicuous part of the wound flora, despite some observations questioning their existence in wounds. The Clostridia in the soil were extremely varied; and 25 species were recognized among 243 strains, with several unclassified strains. All soil samples showed Clostridia. There was no significant difference between the species of anaerobes isolated from soil in rice paddies, lowlands, and hillsides. An unusual fact was the total absence of *Cl. tetani* in soil; this species was readily isolated from tissues, and it was evident that the organism was extremely rare in the soil of Korea.

During the Korean conflict, fresh wounds were frequently contaminated with a diverse Clostridia. Pathogenic species were less often encountered, however, than in reports of World War II. The effect of time lapse on this factor was obvious. Wounds serious enough to require culture at a general hospital would logically be more apt to harbor Clostridia.

Summary

Two groups of patients, totaling 108, were observed for Clostridia of recently incurred wounds by culture of tissue samples at initial débridement within a few hours after injury.

Contamination by Clostridia was relatively frequent during the summer (84.8 per cent of the cases studied), but less frequent during the winter (57.3 per cent).

Cl. perfringens and *Cl. sporogenes* were the principal Clostridia isolated from wounds. Investigators observed a total of 21 species, including eight pathogenic types.

Clostridial bacteremia occurred in a total of 14 cases (19.4 per cent) of the 71 cases on which blood cultures were obtained. The incidence of this bacteremia varied with the two series, but it was far higher in the summer series than in the winter series.

In the summer series, aerobic flora was predominantly *Enterobacteriaceae*, while the majority of the organisms present in the winter series were streptococci and staphylococci. *Bacillus* species were prominent in both series.

Soil cultures showed a predominance of *Cl. perfringens*, in contrast to skin swabs from which the most frequently recovered strain was *Cl. sporogenes*. In the distribution of Clostridial species, clothing samples were similar to those in the soil, while flora on the skin was more like that of the wounds.

References

1. Weinberg, M., and Seguin, P.: *La Gangrene Gazeuse*. Masson, Paris, 1918.
2. Coupal, J. F.: "Pathology of Gas Gangrene Following War Wounds," *The United States Army in the World War, Vol. XII*, pp. 407-567. Medical Department, United States Army, Washington, D. C., 1929.
3. Smith, L. D.: Clostridia in Gas Gangrene. *Bact. Rev.* 13: 233, 1949.
4. Humphreys, F. B.: Formation of Acrolein from Glycerol by *B. welchii*. *J. Inf. Dis.* 35: 282, 1924.
5. Breed, R. S.; Murray, E. G. D.; and Hitchens, A. P.: *Bergey's Manual of Determinative Bacteriology*, 6th edition. Williams and Wilkins Company, Baltimore, 1948.
6. Reed, G. B., and Orr, J. H.: Rapid Identification of Gas Gangrene Anaerobes. *War Medicine*, 1: 493, 1941.
7. Rustigian, R., and Cipriani, A.: The Bacteriology of Open Wounds. *J. A. M. A.*, 133: 224, 1947.
8. Butler, H. M.: Further Bacteriological Studies of Severe *Clostridium welchii* Infection Following Abortion. *J. Obst. Gynec., British Empire*, 50: 105, 1943.
9. Stock, A. H.: Clostridia in Gas Gangrene and Local Anaerobic Infections During the Italian Campaign. *J. Bact.* 54: 169, 1947.
10. MacLennan, John: Personal communication, 1954.

Chapter 18

The Bacterial Flora of Battle Wounds at the Time of Primary Débridement:

A Study of the Korean Battle Casualty*

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This study was carried out at the 46th Surgical Hospital (Mobile Army) on the eastern front in Korea during August and September of 1952 and from December through February of 1953. Its purpose was to describe the bacterial flora of the battle wound at the time of primary surgical intervention.

During the period of this study, the front lines in eastern Korea were stable; hence most casualties received immediate first aid. Large wounds were often covered with a sterile pad before the casualty was transported. At a battalion aid station, wounds were inspected by a medical officer and, when feasible, sterile dressings were applied to large wounds which had not been covered previously. Small wounds were bandaged unless they were too numerous or too widely distributed. From 300,000 to 600,000 units of penicillin and tetanus toxoid were administered routinely. When ambulance evacuation was used, patients were examined at collecting and clearing company levels prior to arrival at the surgical hospital.

The 46th Surgical Hospital was located approximately ten miles behind the front lines. Resuscitation was continued here for variable periods, depending upon the degree of blood loss and the number of casualties awaiting operation. Following resuscitation, the patient was moved to the operating room and anesthetized; and all bandages were removed.

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Methods

Wounds were prepared for débridement by cleansing with a sterile sponge and by pouring a solution of green soap and then water over the wound. The surrounding skin was painted with tincture of merthiolate and covered with sterile towels. Intrathoracic or intra-abdominal wounds were excluded from this study.

Tissue blocks from 0.5 to 1.0 cc. in size were then cut from the skin margins, subcutaneous tissue, and muscle. These were placed directly into a chopped heart-infusion medium (modified Robertson's). The wounds were débrided, followed by irrigation with normal saline solution before applying dressings. Usually patients were held under observation for several days and given penicillin therapy. If their convalescence appeared uncomplicated, they were sent to the rear through regular evacuation channels.

The culture tubes were sealed with Vaseline; and within 5 to 7 days they were sent by air to the Bacteriology Department of the 406th Medical General Laboratory in Tokyo. Twenty control studies demonstrated that the inoculates remained viable when shipped in this manner.

Clostridial isolates were identified and pathogenicities verified by standard, guinea-pig inoculation.¹ *In vitro* sensitivities of anaerobic isolates to Aureomycin, Terramycin, penicillin, and chloramphenicol were performed by the tube-dilution method.² Aerobic organisms were isolated and identified by routine methods.

In a similar study made during the winter of 1952, 21 samples of soil were taken from bunkers on the main line of resistance, from areas forward of the line which were frequented by patrols, from shower points, and from other areas where troops might come in contact with soil. Skin swabs were made of 40 combat soldiers and 35 samples of clothing were taken from wounded troops. All of these were cultured in the manner described.

Results

Anaerobic Flora

A summary of the anaerobic flora is given in Tables 1, 2, and 3. The wounds were tabulated according to size. Wounds of less than 1 cm. in diameter were listed as small; wounds from 1 to 5 cm. in diameter, medium; and wounds greater than 5 cm. in diameter, large.

Table 1 shows that the tissue blocks from 84 per cent of all wounds studied during the summer contained clostridia; and 44 per cent contained pathogenic clostridia. There was no significant correlation between the bacterial flora and the time lag from injury to biopsy; but the larger wounds had a much higher incidence of pathogenic clostridia.

Table 1. The Anaerobic Flora of Tissue Blocks Taken from 42 Wounds of 33 Patients—Summer 1952

Size of Wound	Total Number of Wounds	Wounds Containing		Time Lag—Injury to Biopsy	
		Nonpathogenic Clostridia	Pathogenic Clostridia	Wounds with Clostridia	Wounds Without Clostridia
Small	21	11 (52%)	4 (19%)	5.0 hours	4.5 hours
Medium	8	8 (100%)	3 (37%)	3.7 hours	3.7 hours
Large	13	13 (100%)	10 (76%)	4.8 hours	4.8 hours
Total Average	42	32 (84%)	17 (44%)	4.5 hours	4.3 hours

Table 2. The Anaerobic Flora of Tissue Blocks Taken from 112 Wounds of 69 Patients—Winter 1952–1953

Size of Wound	Total Number of Wounds	Wounds Containing		Time Lag—Injury to Biopsy	
		Nonpathogenic Clostridia	Pathogenic Clostridia	Wounds with Clostridia	Wounds Without Clostridia
Small	29	7 (24%)	6 (21%)	6.2 hours	10 hours
Medium	39	18 (50%)	9 (23%)	9.6 hours	9 hours
Large	44	15 (34%)	9 (20%)	8.0 hours	8 hours
Total Average	112	40 (36%)	24 (21%)	7.9 hours	9 hours

Bacterial Flora at Primary Débridement

Table 2 shows that tissue blocks from 36 per cent of all wounds studied during the winter contained clostridia. In this group of patients, the size of the wound did not appear to be related to the incidence of contamination by clostridia. As in the earlier study, there did not appear to be a significant correlation between the presence of clostridia and the time lag from injury to surgery.

A comparison of these two studies showed that a significantly higher percentage of wounds examined during the summer contained clostridia than did those examined during the winter. The time lag between injury and surgery, however, was somewhat greater in the winter than in the summer.

Tabulation is made by species of the distribution of clostridia in wounds, on the skin and clothing, and in the soil (Table 3). The summer series showed an average of 2.3 strains per wound, while for the winter series it was 1.5 strains per wound.

Table 3. Distribution of Clostridial Species in Wounds, Skin, Clothing, and Soil

Clostridium Species	Percentage of Clostridia				
	Summer 42 Wounds	Winter 112 Wounds	Skin Swabs 40 Samples	Clothing 35 Samples	Soil 21 Samples
<i>Cl. sporogenes</i>	45. 2	8. 9	10	14. 2	30. 9
<i>Cl. perfringens</i>	30. 9	8. 9	10	22. 5	90. 4
<i>Cl. novyi</i>	9. 5	3. 5	5	-----	4. 7
<i>Cl. paraputrificum</i>	9. 5	0. 8	-----	-----	-----
<i>Cl. lentoputrescens</i>	4. 7	3. 5	-----	-----	9. 5
<i>Cl. bifermentans</i>	23. 8	1. 7	-----	-----	23. 8
<i>Cl. sordelli</i>	-----	2. 6	-----	5. 7	-----
<i>Cl. pasteurianum</i>	-----	-----	-----	-----	9. 5
<i>Cl. carnis</i>	-----	3. 5	-----	-----	4. 7
<i>Cl. tetani</i>	-----	1. 7	2. 5	5. 7	-----
<i>Cl. tertium</i>	9. 5	1. 7	2. 5	-----	4. 7
<i>Cl. capitovale</i>	-----	2. 6	2. 5	-----	-----
<i>Cl. fallax</i>	-----	0. 8	-----	5. 7	-----
<i>Cl. putrefasciens</i>	-----	0. 8	2. 5	-----	-----
<i>Cl. tetanomorphum</i>	7. 1	-----	5	-----	9. 5
<i>Cl. parabotulinum</i>	-----	-----	2. 5	5. 7	-----
<i>Cl. butyricum</i>	4. 7	0. 8	-----	11. 4	23. 8
<i>Cl. difficile</i>	-----	-----	-----	-----	4. 7
<i>Cl. aerofetidum</i>	2. 3	-----	2. 5	-----	19. 4
<i>Cl. subterminale</i>	-----	-----	-----	-----	-----
<i>Cl. multifementans</i>	14. 2	1. 7	5	-----	14. 2
<i>Cl. sphenoides</i>	2. 3	0. 8	-----	-----	4. 7
<i>Cl. histolyticum</i>	2. 3	1. 7	-----	-----	-----
<i>Cl. cochlearium</i>	2. 3	0. 8	2. 5	5. 7	-----
<i>Cl. microaerophilus</i>	-----	-----	2. 5	14. 2	23. 8
unclassified	-----	2. 6	5	-----	-----

Pathogenicity of Clostridia

The following organisms were found to be pathogenic in guinea pigs: *Cl. perfringens*, *Cl. carnis*, *Cl. chauvoei*, *Cl. septicum*, *Cl. novyi*, *Cl. sordelli*, *Cl. histolyticum*, *Cl. tetani*, *Cl. difficile*, *Cl. paratubulinum*, and *Cl. fallax*. This pathogenicity compared favorably with those described in the literature.³

Tables 4 and 5 show that sensitivities of 126 clostridial isolates to penicillin, Aureomycin, Terramycin, and chloramphenicol followed normal-distribution curves. Aureomycin and Terramycin appear to be inhibitory in the lowest concentrations. Those concentrations were omitted which were not inhibitory.

Aerobic Flora

The aerobic organisms isolated from the wounds (Table 6) were those commonly found in soil, air, organic matter, and in the respiratory tracts of medical personnel. The tissue blocks from 89 per cent of the wounds cultured during the summer contained pathogenic and nonpathogenic aerobes; and 81 per cent of those cultured in the winter were positive for aerobes. Strains of staphylococcus and streptococcus were found more frequently during the winter months, while the colon group of organisms were found more frequently in summer. The size of the wound did not appear to influence the presence or absence of aerobic organisms.

Discussion

The data presented confirm the belief that a large percentage of battle wounds contain pathogenic aerobes and anaerobes. It is likely that the actual incidence of simple contamination may be higher than presented here, since only isolated blocks from representative portions of any single wound were cultured. It is entirely probable that a considerable number of organisms were missed by this method of examination.

The strikingly lower incidence of clostridial contamination in wounds during the winter months may have resulted from a decrease in the clostridial population found in the environment. Moisture and organic matter are factors necessary for clostridial proliferation.⁴ With freezing temperatures, these growth factors were not present and bacteria remained dormant. Furthermore, direct contact of the troops with the soil may be diminished when the terrain is frozen and covered with ice and snow.

The importance of the presence of aerobes in 89 per cent of wounds which occurred during the summer and 81 per cent during the winter cannot be minimized. Not only do these organisms have the ability

Table 4. Sensitivities of 126 Clostridial Isolates to Penicillin and Aureomycin

Clostridium Species	Num- ber of Species Iso- lated	Penicillin units/cc.*										Aureomycin micrograms/cc.									
		.025	.1	.2	.4	.8	1.6	3.2	6.4	>6.4		.0125	.025	.05	.1	.2	.4	.8	1.2	1.6	>3.2
Cl. capitolale	3	1							1		1						1				
Cl. carnis	2	1			1										1					1	
Cl. cochlearium	5				3		2		1						1	4					
Cl. fallax	1													1				1			
Cl. lentoputrescens	4				1	2	1							2	4	5					
Cl. multi fermentans	10				1		7		1					1	1	1					
Cl. novyi	4						3	1													
Cl. parabo tulinum	1					1								1							
Cl. paraputrificum	2						1	1										1			
Cl. perfringens	23			6	6	4	4		1	2	2	3	3	6	3	3	2			1	
Cl. sordelli	5			1		1	1		2				1	1	2	1					
Cl. sphenoides	1				1											1					
Cl. sporogenes	29			1	5	11	7	2	2	1			10	10		7	1			1	
Cl. tertium	1								1						1						
Cl. tetani	2														1	1	1				
Cl. tetanomorphum	3													1		1	1				
unclassified	30	3	6	7	5	5			4		1	1	2	6	3	10	5	1	1	1	
Total	126	1	3	17	26	25	32	5	5	12	3	4	17	30	17	37	9	4	1	4	

*Penicillin, 1 unit=0.6 microgram.

Table 5. Sensitivities of 126 Clostridial Isolates to Terramycin and Chloramphenicol

Clostridium Species	Num- ber of Spe- cies Iso- lated	Terramycin micrograms/cc.										Chloramphenicol micrograms/cc.									
		.025	.1	.2	.4	.8	1	2	4	6	8	12	16	20	32	>32					
<i>Cl. capitolale</i>	3	1					1														
<i>Cl. carnis</i>	2		1																		
<i>Cl. cochlearium</i>	5			1	2	1	1														
<i>Cl. fallax</i>	1					1															
<i>Cl. lentoputrescens</i>	4		2		5	5															
<i>Cl. multi fermentans</i>	10																				
<i>Cl. novyi</i>	4		1	1	1	2															
<i>Cl. parabolulinum</i>	1																				
<i>Cl. paraputrificum</i>	2																				
<i>Cl. perfringens</i>	23		4	3	6	9															
<i>Cl. sordelli</i>	5		1	1	2	1															
<i>Cl. sphenoides</i>	1				1																
<i>Cl. sporogenes</i>	29		10	9	1	6	1	1													
<i>Cl. tertium</i>	1																				
<i>Cl. tetani</i>	2		1		1	1															
<i>Cl. tetanomorphum</i>	3		2	1	1	1															
unclassified	30		2	4	3	8	2	4	3	1	3	1	3	1	1	1	2	11	6	2	1
Total	126	1	19	23	22	36	5	6	2	6	2	6	9	3	2	2	39	26	18	9	3

Bacterial Flora at Primary Débridement

Table 6. Distribution of Aerobes in Wounds

Microorganism	Percentage of Wounds	
	Summer, 42	Winter, 112
Staphylococcus:		
hemolytic	16. 6	45. 5
nonhemolytic	11. 9	33. 0
Streptococcus:		
alpha	21. 4	25. 7
beta	-----	60. 7
gamma	35. 7	50. 0
<i>Gaffkya tetragena</i>	-----	2. 6
<i>Bacillus</i> species	9. 5	61. 6
<i>Corynebacterium</i> species	4. 7	17. 8
<i>E. coli</i>	21. 4	0. 8
<i>Aerobacter aerogenes</i>	19. 0	0. 8
<i>Paracolon</i> species	11. 9	2. 6
<i>Proteus</i> species	16. 6	6. 2
<i>Pseudomonas aeruginosa</i>	-----	1. 6

to produce severe infections, but they may have an influence on the proliferation or inhibition of other pathogenic anaerobes present which must not be overlooked.⁴ The complex nature of symbiotic relationships between bacteria in wounds requires further investigation. The presence of pathogenic aerobes and anaerobes in both small and large wounds re-emphasizes the possibility of severe infections resulting from the delay in treatment or improper surgical management. The higher incidence of strains of streptococcus and staphylococcus in wounds occurring in the winter may result from the prevalence of upper respiratory infections during this season. The greater frequency of the colon group of organisms seen in wounds occurring in the summer might be explained by the fact that the soil in Korea is fertilized with raw human excrement; and these organisms tend to proliferate in the absence of freezing temperatures.

In vitro sensitivity studies of clostridial isolates to various antibiotics show that lower concentrations of Aureomycin and Terramycin than of penicillin are able to inhibit growth. During the Korean conflict, penicillin was used almost exclusively for prophylaxis of wound infections. Perhaps the re-evaluation of antibiotic therapy after battle injury would lead to a more extensive use of the newer antibiotics.

Similar studies from World War I and World War II have appeared in the literature.⁵⁻⁸ All these studies confirmed the presence

of pathogenic aerobes and anaerobes in wounds prior to initial surgery. Clostridia were usually found in 40 to 50 per cent of wounds, and aerobes in 80 to 90 per cent. Despite the high incidence of simple contamination, the actual occurrence of gas gangrene was strikingly low in most studies. During World War I, Gross and co-workers reported an incidence of 1.9 per cent and 3 per cent in several series including large numbers of wounded soldiers.⁹ During World War II, MacLennan reported anaerobic cellulitis occurring in 1 per cent of the wounded soldiers and obvious gas gangrene in from 0.32 to 0.7 per cent.⁷ Other reviews suggest that there was little difference in the incidence of serious wound infections between the two world wars.¹⁰ It appears that, with early and complete débridement of devitalized tissue and use of antibiotics, the occurrence of serious wound infections was lower during World War II.

In a study carried on during the latter phase of the Korean conflict, Howard and Inui found that the incidence of gas gangrene in 4,900 casualties dropped to 0.08 per cent and mortality to zero.¹¹ This decreased incidence of severe clostridial infection was not due to a decrease in contamination of wounds with pathogenic organisms. It probably resulted from earlier débridement, repair of arterial injuries, and the consistent use of antibiotics.

Conclusions

1. A large percentage of the battle wounds examined within 8 hours after injury contained pathogenic aerobes and anaerobes as contaminants.

2. A smaller percentage of wounds showed contamination during the winter than do those cultured during the summer.

3. *In vitro* sensitivity studies of clostridial isolates to various antibiotics showed that the antibiotics, Aureomycin and Terramycin, were able to inhibit growth in the lowest concentrations.

References

1. Reed, G. B., and Orr, J. M.: Rapid Identification of Gas Gangrene Anaerobes. *War Medicine*, 1: 493, 1941.
2. Newton, A.; Strawitz, J. G.; Lindberg, R. B.; and Howard, J. M.: Sensitivities of Ten Species of Clostridia to Penicillin, Aureomycin, Terramycin, and Chloramphenicol. (Chapter 16 of this volume.)
3. Breed, Robert S.; Murray, E. G. D.; and Hitchens, A. P.: *Bergey's Manual of Determinative Bacteriology*, 6th ed. Williams and Wilkins Company, Baltimore, Maryland, 1948.
4. Ireland, M. W.; Callender, G. R.; and Coupal, J. F.: Pathology of Gas Gangrene Following War Wounds, Sec. II, pp. 407-567, *The Medical Department of the United States Army in the World War, Vol. XII*. U. S. Government Printing Office, Washington, D. C., 1929.
5. Stoddard, J. L.: The Occurrence and Significance of *B. welchii* in Certain Wounds. *J. A. M. A.* 71: 1400, 1918.
6. MacLennan, J. D.: Anaerobic Infections of War Wounds in the Middle East. *Lancet* 245: 123 (July 31), 1943.
7. MacLennan, J. D.: Anaerobic Infections in Tripolitania and Tunisia. *Lancet* 246: 203 (Feb. 12), 1944.
8. Rustigian, R., and Cipriani, A.: The Bacteriology of Open Wounds. *J. A. M. A.* 133: 224, 1947.
9. Gross, Sieru, Mercier, and Lardenois: Treatment of Gas Bacillus Infection. Cited by Van Bouran.
10. Smith, L. D.: Clostridia in Gas Gangrene. *Bacteriological Reviews* 13: 233, 1949.
11. Howard, J. M., and Inui, F. K.: Clostridial Myositis—Gas Gangrene: Observations of Battle Casualties in Korea. *Surgery* 36: 1115, 1954. (Chapter 15 of this volume.)

Chapter 19

The Bacterial Flora of Healing Wounds:

A Study of the Korean Battle Casualty*

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Several studies have described the bacterial flora of battle wounds at the time of initial débridement. In one study during World War I, Stoddard found *Clostridium perfringens* present in 80 per cent of the wounds.¹ Working in the Middle East and later in Tripolitania and Tunisia during World War II, MacLennan found that between 45 per cent and 50 per cent of the wounds contained clostridia.^{2 3} In the Pacific area, Neel and Cole found that 48 per cent of 160 wounds which were cultured shortly after injury contained clostridia.⁴ Except for differences in species distribution, the pattern of simple contamination was similar in wounds of the Korean casualties. At the time of primary débridement, 46 per cent were found to contain clostridia.⁵

This study was undertaken to explore the fate of microorganisms in the open, débrided wound during the earlier stages of healing.

Material and Methods

Patients received their earliest surgical treatment at hospitals located within 10 miles of the main line of resistance.

At battalion aid stations, external hemorrhage was controlled; the wounds were dressed; fluid replacement therapy was initiated; and penicillin (300,000–600,000 units) and tetanus toxoid were given routinely.

When resuscitation was considered optimum, the patient was anesthetized and the area surrounding the wound was cleansed with tincture of green soap and sterile water. The surrounding skin was prepared with tincture of merthiolate, and the surgical field was draped with sterile towels.

During March and April of 1953, 11 patients were selected for study. Selection was based on the accessibility of the wound for serial biopsies

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and on its location somewhat removed from any focus of subsequent contamination. In the majority of patients, the wound of entrance in the skin was small as compared with the extent of destruction of the underlying subcutaneous tissue and muscle. Thus, when a perforating wound was incised, a large cavity was sometimes found in the deeper layers.

The initial surgery of these wounds consisted of débridement of skin, subcutaneous tissue, and muscle. The wounds were then covered with fine-mesh gauze and a thick, sterile dressing. Each patient was given penicillin (300,000 units) and streptomycin (.05 gm.) intramuscularly every 12 hours.

At the beginning of débridement, tissue blocks were taken from healthy muscle in the base of each wound. In addition, blocks of devitalized muscle were taken from six of the wounds. Subsequently, tissue blocks from typical areas of healing muscle were taken at 2-day intervals. For these secondary biopsies, the patients were returned to the operating room where sterile technic was used. After the last tissue block was taken and wound closure accomplished, patients were held from 1 to 3 days before being evacuated.

Each tissue block was placed in a medium of cooked meat broth (modified Robertson's); the tube was sealed with Vaspar and sent by air to the 406th Medical General Laboratory in Tokyo. Twenty control specimens, which were held and shipped in a similar manner, maintained viable organisms.

Isolation and Identification

The incoming specimens were incubated for 24 hours at 37° C. Then 1 ml. was transferred to thioglycollate broth and, in turn, it was incubated for an additional 18 hours at 37° C. A yeast-extract, blood-agar, plate and a chloral-hydrate, sodium-azide plate were streaked with a 4 ml. loop from the thioglycollate subculture and incubated anaerobically for 40 hours. A representative of each morphologic colony was selected from each plate and transferred to thioglycollate broth. After an 18-hour incubation period, gram stains were performed. The gram-positive, spore-forming bacilli were plated on blood-agar base and incubated aerobically. Those that showed no growth on the aerobic plates were replated on blood-agar base and incubated anaerobically for 48 hours to determine the uniformity of colony type. A representative colony was transferred to thioglycollate broth to be used as a seed culture for biochemical identification.

Identification of the aerobic flora was effected by standard laboratory technics. The clostridia were identified by using a modification of the Reed and Orr classification, utilizing 10 carbohydrates, hy-

drogen sulfide production, litmus milk reaction, gelatine liquefaction, indole production, acrolein production, nitrate reduction, and pathogenicity for guinea pigs.⁶

Results

Anaerobes. Nine of the 11 wounds studied contained one or more species of clostridia at the time of débridement (Table 1). One wound did not demonstrate clostridia on initial culture (Patient 1) but showed one species on the fifth day. Clostridia were never isolated from the remaining wound (Patient 10). Two of the wounds (Patients 3 and 7) were clinically infected, but neither showed gas gangrene. Clostridia were absent from two-thirds of the wounds when studied following initial surgery.

Aerobes. All wounds contained a rich flora of gram-positive and gram-negative aerobes on initial culture (Table 1). Without exception, these organisms persisted in the wounds during the first week of healing. However, there appeared to be considerable variation in the number of organisms and species from one culture to the next.

Pathogenicity of Clostridia

By using the standard guinea-pig inoculating procedure, the following species of clostridia were found to be pathogenic in this laboratory: *Cl. perfringens*, *Cl. fallax*, *Cl. carnis*, *Cl. novyi*, *Cl. sordelli*, *Cl. histolyticum*, *Cl. difficile*, *Cl. tetani*, and *Cl. parabolulinum*.⁷ The findings in this study compared favorably with pathogenicity of clostridia described previously.

Discussion

Although this series is small, the consistency of the observations permits valid conclusions. The rich aerobic and anaerobic flora demonstrated in most wounds confirmed the results of similar studies since World War I.

The absence of clostridia in two-thirds of cultures taken subsequent to initial surgery suggests that the present methods of débridement of devitalized tissue are important factors in reducing the bacterial flora.

The two wounds in which adequate débridement was not performed (Patients 3 and 7) showed clinical evidence of infection; they contained rich clostridial and aerobic flora throughout the first week of healing. These findings are consistent with the belief that nonviable tissue is one of the necessary requirements for maintaining clostridia. The persistently rich, aerobic flora in all cultures is not surprising, since these organisms are found extensively in skin, air, soil, and in the respiratory tracts of medical personnel.

Bacterial Flora of Healing Wounds

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*H+, hemo
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Table 1. The Bacterial Flora of Eleven Wounds During the First Week of Healing

Patient	Time-Lag Injury to Surgery	Antibiotic Hours Prior to Surgery	Wounding Agent	1st Hospital Day		3d Hospital Day		4th Hospital Day		5th Hospital Day		6th Hospital Day		7th Hospital Day		8th Hospital Day	
				Anaerobes	Aerobes*	Anaerobes	Aerobes	Anaerobes	Aerobes	Anaerobes	Aerobes	Anaerobes	Aerobes	Anaerobes	Aerobes	Anaerobes	Aerobes
1	4 hrs.	300,000 units penicillin 3.5 hrs.	small arms	none isolated	Strep. beta <i>Corynebact. sp.</i> Staph. H+S+M-C- Staph. H+S+M+C+	none isolated	Strep. beta Strep. alpha Bacillus sp.			<i>Cl. novyi</i>	<i>Ps. aeruginosa</i> Paracolon sp. Strep. alpha Bacillus sp.			not examined			
2	9 hrs.	300,000 units penicillin 7.5 hrs.	shell fragments	<i>Cl. novyi</i>	Strep. H- Bacillus sp.	none isolated	Strep. beta			none isolated	<i>Corynebact. sp.</i> Strep. beta Staph. H+S+M+C+			none isolated	Strep. beta <i>E. coli</i>		
3	6 hrs.	300,000 units penicillin 5 hrs.	small fragment	<i>Cl. perfringens</i> <i>Cl. bifermentans</i> <i>Cl. paraptrophicum</i>	Strep. beta Strep. H- <i>Sarcina lutea</i>	<i>Cl. novyi</i> <i>Cl. perfringens</i> <i>Cl. sporogenes</i>	Strep. beta Strep. H- Bacillus sp. <i>E. coli</i> Strep. alpha			<i>Cl. sporogenes</i> <i>Cl. bifermentans</i>	Strep. H- Strep. alpha Bacillus sp.	<i>Cl. perfringens</i>	Strep. H- Strep. alpha			<i>Cl. novyi</i> <i>Cl. sporogenes</i>	Strep. H-
4	2 hrs.	600,000 units penicillin 2.5 hrs.	small fragment	Devitalized Muscle: <i>Cl. bifermentans</i> <i>Cl. novyi</i> <i>Cl. multimentans</i> <i>Cl. sporogenes</i> <i>Cl. paraptrophicum</i> Muscle After Débridement: <i>Cl. tetanomorphum</i> <i>Cl. sporogenes</i>		<i>Cl. sporogenes</i> <i>Cl. bifermentans</i>	none isolated			<i>Cl. sporogenes</i> <i>Cl. bifermentans</i> <i>Cl. multimentans</i> <i>Cl. butyricum</i> <i>Cl. carnis</i>	Bacillus sp.			<i>Cl. novyi</i>	Staph. H+S+M+C- Strep. H- Bacillus sp.	not examined	
5	10 hrs.	no record	mortar fragments	Devitalized Muscle: <i>Cl. perfringens</i> Muscle After Débridement: none isolated		none isolated	Strep. beta Strep. H- Bacillus sp. Staph. H+S+M+C-					none isolated	Bacillus sp.			none isolated	Staph. H+S+M+C-
6	6 hrs.	300,000 units penicillin 4 hrs.	shell fragments	Devitalized Muscle: <i>Cl. tetani</i> <i>Cl. sporogenes</i> <i>Cl. novyi</i> <i>Cl. paraptrophicum</i> <i>Cl. tetanomorphum</i> Muscle After Débridement: <i>Cl. butyricum</i>		none isolated	Bacillus sp.					none isolated	Bacillus sp.			none isolated	Strep. H- <i>Ps. aeruginosa</i>
7	6.5 hrs.	300,000 units penicillin 3.5 hrs.	shell fragments	<i>Cl. perfringens</i> <i>Cl. sordelli</i> <i>Cl. butyricum</i> <i>Cl. bifermentans</i>	Strep. H- Bacillus sp.	<i>Cl. sordelli</i>	<i>A. aerogenes</i>					<i>Cl. perfringens</i> <i>Cl. bifermentans</i> <i>Cl. aerofetidium</i> <i>Cl. butyricum</i>	<i>E. coli</i>			none isolated	Strep. beta H+ Bacillus sp. <i>Serratia marcescens</i>
8	6 hrs.	300,000 units penicillin 5 hrs.	land mine	<i>Cl. paraptrophicum</i>	Staph. H+S+M-C- Bacillus sp. Paracolon sp.	none isolated	Strep. beta Strep. H- Staph. H-S+M+C-			none isolated	Strep. beta Staph. H+S-M-C- Bacillus sp. Strep. H-			none isolated	Strep. beta Staph. H+S+M+C-		
9	6 hrs.	300,000 units penicillin 5.5 hrs.	band grenade	<i>Cl. carnis</i> <i>Cl. butyricum</i>	Bacillus sp. Strep. H-			none isolated				none isolated	Bacillus sp.			none isolated	Staph. H+S+M-C- Bacillus sp. Staph. H+S+M+C+ Staph. H-S+M+C+
10	4 hrs.	no record	shell fragments	Devitalized Muscle: none isolated				none isolated	Bacillus sp. Strep. H-			none isolated	Staph. H+S+M+C-			9th Hospital Day	
				Muscle After Débridement: none isolated												Anaerobes	Aerobes
				Muscle After Débridement: none isolated												none isolated	Staph. H+S+M+C- Bacillus sp. Strep. H-
11	3 hrs.	300,000 units penicillin 2.5 hrs.	mortar fragments	Devitalized Muscle: <i>Cl. sporogenes</i> <i>Cl. lentoputrescens</i> Muscle After Débridement: none isolated		none isolated	Strep. alpha Strep. H- Bacillus sp. <i>Corynebact. sp.</i>			none isolated	none isolated			none isolated	Bacillus sp.		

No.	Name	Age	Sex	Date
1	John Smith	25	M	1910
2	Mary Jones	22	F	1910
3	James Brown	28	M	1910
4	Elizabeth White	24	F	1910
5	Robert Black	26	M	1910
6	Sarah Green	23	F	1910
7	William Hall	27	M	1910

The changing character of both the aerobic and the clostridial flora from one hospital day to another was observed in all wounds. Further contamination at wound dressing could account for the appearance of many new aerobic organisms. Antibiotic therapy may have influenced both the aerobic and the anaerobic flora, but it did not influence appreciably the flora in the presence of necrotic tissue. Culturing a single, small-tissue block from a large wound probably did not give a complete picture of the total bacterial flora. The consistency of the findings, however, suggests that a representative index of the bacterial flora was obtained.

Histologic studies of the same wounds are reported in another paper.⁸ In the absence of necrotic tissue, the bacterial flora did not appear to effect the healing of the open wound.

Conclusions

1. Pathogenic and nonpathogenic clostridia were isolated in 9 of 11 wounds at the time of initial surgery. All wounds contained a rich aerobic flora.

2. Only three of the four wounds showing clostridia at the time of initial surgery contained these organisms in subsequent cultures. This would appear to indicate that present methods of débridement and irrigation are effective in reducing the bacterial population in wounds.

3. At the time of secondary closure, pathogenic aerobic organisms are often present in the wound. In the absence of other deterrents, however, they show no clinical evidence of interfering with wound healing.

References

1. Stoddard, J. L.: The Occurrence and Significance of *B. welchi* in Certain Wounds. *J. A. M. A.* 71: 1400, 1918.
2. MacLennan, J. D.: Anaerobic Infections of War Wounds in the Middle East. *Lancet*, 245: 123, 1943.
3. MacLennan, J. D.: Anaerobic Infections in Tripolitania and Tunisia. *Lancet*, 246: 203, 1944.
4. Neel, H. B., and Cole, J. P.: Gas Gangrene in Amphibious Warfare in the Pacific Area. *Am. J. Surg.* 66: 290, 1944.
5. Lindberg, R. B.; Wetzler, T. F.; Marshall, J. D.; Newton, A.; Strawitz, J. G.; and Howard, J. M.: The Bacterial Flora of Battle Wounds at the Time of Primary Débridement: A Study of Korean Battle Casualties. *Ann. Surg.* 141: 369, 1955. (Chapter 18 of this volume.)
6. Reed, C. B., and Orr, J. H.: Rapid Identification of Gas Gangrene Anaerobes. *War Med.* 1: 493, 1941.
7. Breed, R. S.; Murray, E. G. D.; and Hitchens, A. P.: *Bergey's Manual of Determinative Bacteriology*, 6th edition. Williams and Wilkins Company, Baltimore, Maryland, 1948.
8. Scully, R. E.; Artz, C. P.; and Sako, Y.: The Criteria for Determining the Viability of Muscle in War Wounds. (Chapter 13 of this volume.)

Chapter 20

The Bacterial Flora of the Blood in the Korean Battle Casualty*

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The purpose of this study was to describe the bacterial flora of the blood stream of the severely injured casualty at the time of his arrival at the 46th Mobile Army Surgical Hospital. The primary intent was to describe the bacterial flora for guidance in therapy. A secondary purpose was to survey this problem in patients in deep shock in order to ascertain the possible secondary development of bacteremia due to permeation of the ischemic bowel.¹⁻³

The importance of this information lies in the fact that many casualties had large contaminated wounds of soft tissues; and others had perforations of the gastrointestinal tract with peritoneal contamination.

This study was carried out on the eastern front in Korea from January 1952 through June 1953. At this time, the tactical conditions were stable to such an extent that evacuation of casualties were optimal. Except when wounded on patrol, the casualty usually reached a battalion aid station within 1 hour after injury. At the aid station, antibiotics were often administered in addition to the vital resuscitative measures; however, such measures were less frequently employed by medical officers of the Korean Army. The treatment most frequently given was 600,000 units of penicillin, intramuscularly. The severely injured casualty was evacuated immediately by helicopter or ambu-

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lance. At the time of arrival at the forward surgical hospital, the casualty was taken immediately to the Resuscitation Ward. Blood cultures were obtained at this time on only the most severely wounded casualties.

This study includes only the severely wounded casualties as they were admitted to the forward hospital. The average time following injury was 3.5 hours.

Methods and Materials

After preparing the skin with iodine and alcohol, venous blood was drawn using aseptic technic. For aerobic culture, from 1 to 3 cc. of blood were added to 7 cc. of dextrose broth. For anaerobic culture, 5 cc. of blood were added to a 100-cc. bottle containing 50 cc. of trypticase soy-bean broth and carbon dioxide at a subatmospheric pressure (Becton-Dickinson Vacutainer Bottle). In the first study, penicillinase was added to all media at the time of inoculation. After an incubation period of 48 hours or longer, smears were examined and, at the same time, blood-agar plates and tubes of cooked meat broth were inoculated. The latter were incubated 72 hours, then subcultured for recovery of clostridial organisms. The original culture tubes were retained for a period of 1 or 2 months and checked for delayed growth.

A study of 170 patients was carried out at two separate laboratories. In the first group, 117 casualties were studied; and, of this number, 35 patients had received penicillin prior to culture. Of the group receiving penicillin, 8 patients had also received 1.0 gm. of streptomycin. In the second group, 52 patients were studied; and, of this number, 37 patients had received penicillin and four had also received streptomycin. The time lag was similar in the two studies.

In this study, patients were considered in shock if they presented signs of blood-volume deficiency in association with a systolic blood pressure below 100 mm. and a diastolic below 70 mm. If these signs were associated with a systolic blood pressure below 50 mm., the patient was considered to be in severe shock. Accordingly, in the first series, 59 patients were in shock on admission to the hospital, and 26 of these patients were in deep shock. Twenty-eight patients had visceral perforations. In the second group, 31 patients were in shock and 12 of this number were in deep shock. In the second series, 16 patients had visceral perforations. The two groups, therefore, consisted of casualties with comparable degree of injury. Twenty normal soldiers served as controls. In addition, 20 contaminated cultures were sent from the forward surgical hospital to the base laboratory, where they were recultured to establish the feasibility of shipment.

The Battle Wound: Clinical Experiences

Results

Of the 20 contaminated controls, all contained viable bacteria after shipment. Of the 20 control blood-cultures from the normal soldiers, all were negative.

The results of the two studies of battle casualties were similar (Table 1). In the first study of 117 casualties, 12 cultures were positive (10 per cent). The predominant organisms grown were *Staphylococcus albus* and the organisms of the *Bacillus* species which are among the chief organisms of the skin and the air respectively (Table 2). It is entirely conceivable that several of these positive cultures were the result of contamination, as it was sometimes difficult to obtain blood from a patient who was in shock.

Table 1. The Bacterial Flora of the Blood

Series	Number of Patients	Number of Positive Cultures	Percentage of Positive Cultures
1	117	12	10
2	53	11	21
Total Average	170	23	13.5

Table 2. Organisms Isolated from Blood Culture—Series 1

Patient	Aerobic	Anaerobic
A	<i>Staphylococcus albus</i> , nonhemolytic	negative
B	<i>Staphylococcus albus</i>	negative
C	<i>Bacillus</i> species	negative
	<i>Staphylococcus albus</i>	
D	<i>Bacillus</i> species	negative
E	<i>Bacillus</i> species	negative
F	<i>Staphylococcus albus</i>	negative
G	<i>Staphylococcus albus</i> , hemolytic and nonhemolytic	negative
H	<i>Staphylococcus albus</i>	negative
I	<i>Staphylococcus</i> , hemolytic	negative
J	<i>Corynebacterium hofmanni</i>	negative
	<i>Bacillus</i> species	
	<i>Staphylococcus</i> , hemolytic	
K	<i>Streptococcus</i> , gamma	negative

The results of the second series differed in that Streptococcal strains were cultured from four casualties and clostridia were cul-

Bacterial Flora of the Blood

Table 3. Organisms Isolated from Blood Culture—Series 2

Patient	Aerobic	Anaerobic
A	Staphylococcus, nonhemolytic <i>Corynebacterium hofmanni</i>	negative
B	Staphylococcus*	negative
	Bacillus species	
C	Staphylococcus, nonhemolytic	negative
D	Bacillus species	negative
E	Bacillus species	negative
F	<i>Achromobacter</i>	negative
G	Streptococcus, beta hemolytic	negative
H	Streptococcus, beta hemolytic	negative
	Streptococcus, nonhemolytic	
I	Streptococcus, nonhemolytic	negative
	Staphylococcus**	
J	Streptococcus, nonhemolytic	negative
K	Staphylococcus**	<i>Clostridium sporogenes</i>
	Negative	<i>Clostridium sporogenes</i>
	Negative	<i>Cl. lentoputrescens</i>

*Hemolytic, salt-resistant, mannite-fermenting, coagulase-negative.

**Hemolytic, salt-resistant, mannite-nonfermenting, coagulase-negative.

tured twice.* Coliform organisms were found only once. The incidence of positive cultures was 21 per cent (Table 3).

The incidence of positive cultures could not be related to the time lag, degree of shock, visceral perforation, or previous administration of antibiotics.

Discussion

In the over-all study of 170 severely injured battle casualties, 23 positive blood cultures were found (13.5 per cent). In spite of careful technic, several of these may have represented contamination from the skin or the air; hence the figure is maximal. It is believed that the actual incidence of positive cultures of the peripheral blood stream lies between 4 and 6 per cent in the most severely injured battle casualties; and presumably the wound is the portal of entry. Vascular collapse did not appear to alter this incidence. The results of this study in no way invalidate the observations of other investigators regarding the role of bacteria in the late refractory shock of animals.¹⁻³ However, of the 30 patients studied who died

*Prior to our study, this observation of a clostridial bacteremia was noted repeatedly in cases which were unreported and in a patient with peritonitis. No obvious break in technic was noted, but contamination can never be completely excluded.

as a result of wounds, only three had positive cultures. Moreover, the 38 patients were in severe shock for an average of 7.3 hours (ranging from 1 to 36 hours). Cultures were positive for only three. One of these had been in shock 2 hours, and the other two for 4 hours.

Summary

A study of the bacterial flora of the blood stream in the severely injured battle casualty was carried out by two laboratories, one in Korea and the other in Japan. Blood cultures were studied on 170 severely injured casualties at the time of admission to a forward hospital. The incidence of positive cultures was 13.5 per cent. Correction for possible contamination might lower this incidence as much as 4 or 6 per cent.

The degree of shock, the type of injury, the time-lag, and the previous antibiotic therapy did not affect the incidence of positive cultures.

References

1. Fine, J., and Frank, H. A.: Progress Report, September 15, 1952. Subcommittee on Shock, National Research Council, Washington, D. C.
2. Fine, J.; Frank, H. A.; Schweinburg, F. B.; Jacob, S.; and Gordon, T.: The Bacterial Factor in Traumatic Shock. *Ann. N. Y. Acad. Sci.* 55: 429, 1952.
3. Hardy, E. G.; Morris, G. C., Jr.; Yow, E. M.; Haynes, B. W., Jr.; and De Bakey, M. E.: Studies on the Role of Bacteria in Irreversible Hemorrhagic Shock in Dogs. *Ann. Surg.* 139: 282, 1954.

Chapter 21

A Review of Postmortem Examinations in Combat Casualties*

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Because of the clinical responsibilities of medical officers in a combat theater, complete postmortem examinations have seldom been routine. Pathologists are rarely available at a surgical hospital and frequently facilities for postmortem study are not available. Although it is obvious that combat casualties die from wounds received in action, the mechanisms of death are not clear.

During a 10-month period in 1952 and 1953, autopsies were performed routinely by the Surgical Research Team in Korea at the 46th Surgical Hospital on the eastern front. Deaths were investigated which resulted from wounds received in combat, vehicular accidents, burns, medical illnesses, and self-inflicted injuries.

This report deals only with 35 patients who died in the hospital after being wounded in combat. Analyses of the other traumatic deaths have been deleted in an effort to emphasize the problems encountered in treating the combat casualty. Because of the relatively small number of autopsies presented, statistical significance cannot be ascertained; however, definite trends can be observed and some generalizations can be made.

Most of the autopsies were performed by the same prosector. Clinical histories were recorded in considerable detail and frequently the prosector had observed the clinical course of the patient prior to death. Therefore, it was possible to be consistent in both the examination and the interpretation of results. Microscopic review was carried out by several members of the Pathology Department of the 406th Medical General Laboratory.

Table 1 summarizes the causes of death in 35 combat casualties. There is considerable overlapping between the first three groups: irreversible shock, vital organ damage, and uncontrolled hemorrhage. Some patients having vital organ damage and others having uncon-

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trolled hemorrhage showed changes consistent with those seen in irreversible shock. On the other hand, a few classified as irreversible shock exhibited uncontrolled hemorrhage during some period of their clinical course. Despite this overlapping, the chief cause of death, as determined by evaluating all known factors, formed the basis of this classification. The causes of death in the remaining patients were quite definite.

Table 1. Causes of Death in 35 Combat Casualties

Cause of Death	Number of Patients
Irreversible shock	11
Vital-organ damage	9
Uncontrolled hemorrhage	6
Bronchial obstruction	2
Cardiac arrest	1
Myocardial infarction	1
Blast injury (lungs)	1
Fat embolism (cerebral)	1
Post-traumatic renal insufficiency*	1
Subdural hematoma	1
Cause undetermined	1
Total	35

*Most of the patients having this syndrome were evacuated to the Renal Insufficiency Center; therefore, death rarely occurred at a forward hospital.

Irreversible Shock

The clinician attending six of these patients observed that hemorrhage had been controlled. After operation, however, the patients remained refractory to massive transfusions. These six patients died in a state of unreversed, recurrent shock.

An additional five patients—after prolonged, profound shock—demonstrated a gradual response to transfusion, but died shortly thereafter. The histologic picture in these two groups was similar. Although the clinical course of the latter five patients was not marked by as profound a terminal hypotension as the first group, the autopsy findings suggested that death was due to irreversible ischemic changes secondary to the initial shock.

Table 2 gives the mean for the evacuation time, survival time, and blood-volume replacement in the 11 patients having irreversible shock.

The morphologic changes in irreversible shock which were found in various combinations were: (1) petechial hemorrhages—serosal, mucosal gastrointestinal); (2) dilation and engorgement—vessels in abdominal and thoracic viscera, brain, striated muscle; (3) pul-

Table 2. Mean of Evacuation Time, Survival Time, and Blood-Volume Replacement in Eleven Patients Having Irreversible Shock

Category	Hours	Volume (liters)
Injury to admission	4. 75	
Injury to death	17. 75	
Total replacement		9. 5

monary edema; (4) pulmonary atelectasis—focal and diffuse; (5) dilatation of cardiac chambers with flabbiness of the myocardium; (6) renal tubular changes consistent with post-traumatic renal insufficiency (microscopic); (7) lipid depletion—adrenal cortical cells (microscopic); and (8) fatty vacuolization—heart muscle, liver, kidney (microscopic).

Petechial hemorrhage involving serous surfaces and mucous membranes were frequent and were believed to be the result of prolonged hypoxemia. Subendocardial hemorrhages were often seen, being peculiarly limited to the right side of the heart. This distribution was also noted in patients who died of hemorrhagic fever. It may be that the differences in anatomy and physiology between the left and right heart explain this peculiar localization of hemorrhages; but to date no positive evidence is available.

A consistent finding was intense congestion of all organs and striated muscles. Fluid blood virtually poured out from the cut surface of the lungs, liver, spleen, and kidneys.

Microscopic examination confirmed the severe dilation and congestion of all blood vessels. The large quantity of blood transfused presumably was needed to maintain an adequate arterial pressure because of an expanded vascular tree.

Table 3 shows various degrees of pulmonary edema in the patients having irreversible shock. The majority of these patients demon-

Table 3. Degree of Pulmonary Edema as Compared with Blood-Volume Replacement in Patients Having Irreversible Shock

Degree of Pulmonary Edema	Average Blood-Volume Replacement	Number of Patients
None (below 500 gm.)	2.0 liters	1
Slight (500-700 gm.)	4.8 liters	3
Moderate (700-900 gm.)	12.0 liters	1
Marked (more than 900 gm.)	13.0 liters	6

strated some degree of edema, and over half exhibited marked edema. There appeared to be more edema in those patients who were most vigorously resuscitated. It must be emphasized again that these patients represent a very small sampling and that most patients requiring large transfusions survived.

It has been suggested that pulmonary edema is a constant and integral part of the pathogenesis of shock.¹ Several factors may influence its development. The present study suggests that it follows shock and transfusion rather than ischemia alone. It is a frequent complication of post-traumatic renal insufficiency following shock. Cardiac failure must play a considerable role in the formation of pulmonary edema, as evidenced by gross flabbiness of the myocardium, dilatation of cardiac chambers, and fatty degeneration of myocardial fibers. In a review of a large number of traumatic deaths,² fat embolism did not predispose to this complication.

Varying amounts of pulmonary atelectasis were seen in several patients having irreversible shock. In some lungs, the areas of atelectasis were patchy, while in others the greater part of a single lobe was involved; this was usually basilar in distribution. These pulmonary findings may have represented agonal changes. In one of the 11 patients, diffuse atelectasis was thought to be principal factor contributing to the fatality.

Congestion of striated muscle is listed as a morphologic finding in irreversible shock. This is particularly true of casualties resuscitated with a considerable amount of whole blood and fluids. Striated muscle composes a large part of the human body; but it is seldom studied at the postmortem table. A great part of the vascular tree, however, is found in these large masses of muscle. A typical finding in examining large, avulsive wounds was severe congestion with outpouring of blood and fluids at autopsy incision. Further studies are necessary to correlate this observation with the theory of blood sludging and pooling.

Renal damage was a frequent finding in irreversible shock. The principal morphologic changes consisted of heme casts within tubules and varying degrees of degeneration of the tubular epithelium. These changes, when present in a larger series of patients, were found to be demonstrable after the first 24 hours of shock.³ In the present study, each patient demonstrated a decrease in renal function clinically; however, this condition might have been on a functional rather than on an organic basis. Four of the 11 patients showed morphologic changes consistent with post-traumatic renal insufficiency. The remaining patients might also have exhibited these changes if they had survived longer than the mean of 17.5 hours.

In studying postmortem material from shocked patients during World War II, Mallory found a peculiar morphologic change in the viscera when the survival time was over 18 hours.³ With special stains, fat vacuolization was demonstrated in the heart, the central cells of hepatic lobules, and the ascending limbs of Henle's loops. The doubly refractile lipid became depleted in the adrenal gland after the same time interval. From the fourth day onward, a tendency to a return to normal could be demonstrated in patients with wounds not complicated by infection.

Special stains were not performed in patients presented in this report. However, Mallory's finding is another morphologic demonstration of an intracellular response to injury and shock.

Vital Organ Damage. Nine patients died of damage to vital organs to a degree incompatible with life. Seven showed extensive brain damage, and two had direct cardiac involvement by shell fragments. The majority of these patients survived for several hours.

Uncontrolled Hemorrhage. Table 4 gives information concerning six patients whose deaths were attributed to uncontrolled hemorrhage. All showed profuse bleeding from lacerated, large vascular channels. It appeared in most instances that, if early control of profuse hemorrhage could have been achieved, death might have been prevented. The average evacuation time from injury to admission to a hospital was considerably shorter than that seen in irreversible shock. Pulmonary edema was found less frequently.

Bronchial Obstruction. Two patients died of bronchial obstruction. One patient had a simple amputation of a lower extremity which was technically uncomplicated. He died 15 minutes following operation due to aspiration of vomitus. The second patient suffered a perforating wound of the lung with bleeding into the bronchial tree. Obstruction and death occurred shortly after admission.

Cardiac Arrest. One patient with multiple avulsive and perforating wounds of all extremities expired unexpectedly during operation. Cardiac massage was performed, but without benefit. A moderate number of fat emboli were visible microscopically in small myocardial blood vessels. It is not known what influence they may have had on cardiac function.

Myocardial Infarction (Traumatic Coronary Thrombosis). In one patient, myocardial infarction resulting from coronary thrombosis was a surprising finding. Following a wound of the left lung from small arms fire, this patient developed progressive cardiac failure during operation. At autopsy, an ante-mortem thrombus was found in the lumen of the circumflex branch of the left coronary artery. Histologically, the myocardium exhibited degenerative changes. Although there was no myocardial laceration in the region of the left coronary artery, diffuse subepicardial and perivascular hemorrhages

Table 4. Patients Having Uncontrolled Hemorrhage

Pa- tient	Evacuation Time Injury to Hospital	Total Blood-Volume Replacement	Pulmonary Edema	Site of Bleeding	Survival Time After Injury
1	5.5 hours	10.5 liters	none	laceration of pulmonary vessels	11.8 hours
2	3.0 hours	0	none	splenic rupture, laceration of lung	3.0 hours
3	2.5 hours	total unknown (3.5 liters)	none	laceration of kidney, retroperitoneal hemorrhage	18.0 hours
4	4.0 hours	18.0 liters	none	lacerated vena cava	17.0 hours
5	1.0 hour	6.5 liters	moderate	lacerated dural vessels	4.5 hours
6	1.5 hours	9.5 liters	moderate	multiple lacerations of mesenteric vessels	3.5 hours

suggested that contusion and arteritis may have occurred from the marked pressure changes when the missile passed through the thorax.

Fat Embolism. Fat embolism has been recognized for many years as a complication of trauma and fractures. Different investigators have ascribed varying degrees of importance to it as a clinical entity. Scully showed that fat emboli were present in the lungs in over 90 per cent of the men dying of battle wounds. Occasionally emboli were found in the kidneys. Death resulting from fat embolism was infrequent in his review.²

The criteria for establishing fat embolism as a cause of death are a typical clinical history, abnormal neurologic findings, and the presence of a moderate to a marked degree of fat in the vessels of the lung, kidney, and brain. Parivascular hemorrhages and necrosis in the brain are the usual findings in fatal instances.

The one fatality due to fat embolism presented a classical picture. The patient suffered traumatic amputations of several extremities and numerous avulsive and puncture wounds. Preoperative resuscitation and surgery were uneventful. Recovery, however, from anesthesia was not complete and intracranial hemorrhage was suspected as the patient remained unconscious and displayed muscular twitchings. A craniotomy showed no evidence of hemorrhage. Death ensued several days after injury. Postmortem examination revealed the typical morphologic findings of fatal fat embolism.

Blast Injury. In World War I and World War II, it was observed that injuries to the thoracic and abdominal viscera and to the central nervous system resulted from rapid changes in the environmental pressure, for instance, from an air blast.⁴ When persons are exposed to the effects of nearby shell or mortar explosions, death may occur with minimal evidence of external injury. Hemorrhages and lacerations may occur in the lungs, abdominal viscera, or brain. One patient in this series was close to an exploding mortar round and died shortly afterwards in severe respiratory distress with hypoxia. Autopsy showed that he had severe pulmonary hemorrhage, congestion, contusion, and edema without perforation of the thorax. A large shell fragment was found in the region of the left kidney.

Post-traumatic Renal Insufficiency. One patient having marked renal insufficiency resulting from multiple perforations of the small intestine was included in this series. The patient died from uremia 6 days following injury. The morphologic findings were compatible with severe, lower nephron nephrosis. There were numerous heme casts in renal tubules and marked degeneration of the tubular epithelium. A fibrinopurulent peritonitis was also present.

Subdural Hematoma. In one instance, a subdural hematoma resulted as a complication of a skull fracture. A large subdural blood

clot was removed. Death followed shortly thereafter from increased intracranial pressure.

Cause Undetermined. The one patient in whom a cause of death could not be determined suffered a gunshot wound of the abdomen with a laceration of the tail of the pancreas. His postoperative course seemed uneventful and he was ambulated early. On the sixth postoperative day, he suddenly developed severe respiratory distress and died 12 hours later in vascular collapse. The clinical history was suggestive of pulmonary embolism, but the autopsy did not confirm the diagnosis. There were no significant findings to establish the cause of death.

Summary

An analysis was made of 35 postmortem examinations in combat casualties dying a short time after injury. Irreversible shock, vital organ damage, and uncontrolled hemorrhage constituted the major causes of death. Bronchial obstruction, cardiac arrest, traumatic coronary thrombosis, blast injury, and subdural hematoma were found less frequently. Although fat embolism was found frequently in the lungs following traumatic death, it appeared to be the principal factor causing death in only one patient. Post-traumatic renal insufficiency was the cause of death in only one patient in this series, as patients with this complication were evacuated to another hospital for treatment.

References

1. Moon, V. H.: Shock: Its Mechanism and Pathology. *Arch. Path.* 24: 642 and 794, 1937.
2. Scully, Robert E.: Fat Embolism in Korean Battle Casualties: An Analysis of Its Incidence, Clinical Significance, and Pathologic Aspects. (Chapter 22 of this volume.)
3. Mallory, Tracy B.: The Physiologic Effects of Wounds: Surgery in World War II. (Chairman of the Board, Henry K. Beecher, *Editor*.) The Board for the Study of the Severely Wounded, North African-Mediterranean Theater of Operations. Office of The Surgeon General, Department of the Army, Washington, D. C., 1952.
4. Zuckerman, S.: Experimental Study of Blast Injuries to Lungs. *Lancet* 2: 219, 1940.

Chapter 22

Fat Embolism in Korean Battle Casualties:

An Analysis of Its Incidence, Clinical Significance, and Pathologic Aspects

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The significance of fat embolism following trauma has remained controversial despite the large volume of literature on the subject. Some authors in this field ^{4 5 12 15} have regarded this phenomenon as a relatively common and serious complication of injury, while others ^{3 6 16 17} have felt that it is rarely severe enough to be of clinical importance. Surgeons who handled large numbers of battle casualties in the Korean conflict were seldom able to recognize the clinical syndromes described in the literature as characteristic of fat embolism.^{2 8}

Because of such conflicting opinions, a study of this subject was initiated in the Pathology Section of the 406th Medical General Laboratory in Tokyo, Japan. Autopsy cases of all types of trauma, including that from battle injuries, were analyzed from a clinico-pathologic viewpoint in an attempt to determine the specific significance of fat embolism among the many sequelae of serious injury. This chapter is concerned primarily with the 110 cases of fatal battle casualties reviewed at the laboratory during 1953. Whenever pertinent, data obtained from analysis of similar cases reviewed in 1952 and of cases of death following other types of trauma are presented.

Material and Methods

In 1953, 132 cases of fatal battle injury were studied at our laboratory. Of these, 110 were selected for investigation of the incidence, clinical aspects, and pathologic changes of fat embolism. The remaining 22 cases were eliminated either because clinical or pathologic data were inadequate or because death occurred later than 4 weeks following injury. This arbitrary time limit for exclusion of cases was selected because, in our experience, fat embolism has not been demonstrable beyond this time. Although a few of the soldiers in the series were killed instantaneously or died while being evacuated, the great majority expired at army hospitals where autopsy facilities were

available. Obviously, the 110 studied were primarily those who survived to reach the hospital and do not represent a fair, over-all sample of soldiers who died of battle wounds.

Forty-four of the autopsies were performed by two trained pathologists at the 11th Evacuation Hospital, where casualties were transferred who developed post-traumatic renal failure. A medical officer, who had previous training in pathology, performed 30 autopsies at the 46th Mobile Army Surgical Hospital. The remainder were carried out by various medical officers, some of whom had little experience in pathology, at numerous surgical, evacuation, and general hospitals of the Army either in Korea or in Japan. Moderately detailed clinical abstracts were available on most of the patients autopsied at the 11th Evacuation Hospital and the 46th Surgical Hospital. On the remainder of the patients, the clinical data forwarded to the laboratory were variable, at times being detailed and at other times meager.

Routine autopsy sections of tissue fixed in formalin, embedded in paraffin and stained with hematoxylin and eosin, were available for study. In some instances, only several sections had been prepared; for the great majority of autopsies, however, there were 20 or more sections. In 89 cases, Oil-Red-O fat stains were made on frozen sections of formalin-fixed lung tissue cut at 30 to 40 micra in thickness. In 33 cases, samples of kidney, and in 24, samples of one or several portions of the brain were similarly stained.

All Oil-Red-O sections were graded according to their content of intravascular fat. This grading was attended by some difficulty since the fat was sometimes distributed unevenly in a section. Because it was not feasible to do large numbers of fat stains on any one organ, estimation of the amount of intravascular vacuolation in routine hematoxylin and eosin sections (in which the fat is dissolved out) was resorted to as an ancillary method of grading.

The amount of fat in the lungs was graded: 0 to 8, Grade 1 representing less than 10 small-sized emboli per section, and Grade 8 signifying the presence of emboli in the majority of high-power fields and the appearance of extensive beading of alveolar capillaries with globules of fat. The intermediate grades were difficult to define accurately, however, and selection was made on the basis of visual impressions. Since all sections were examined at least twice and were finally reviewed within a short period of time, it is believed that their grading was reasonably accurate. Grades of Oil-Red-O and of hematoxylin and eosin sections of lung, determined independently of one another, were identical in 47 per cent of the cases; they differed by one in 33 per cent of the cases, by two in 17 per cent, and by three in 3 per cent. When grades differed by one, the higher of the two was selected for statistical purposes; but when the difference was two or three, the average was chosen.

The renal content of fat was graded 0 to 8. Grade 1 corresponded to less than 5 emboli per section, and Grade 8 to the presence of multiple emboli in the great majority of glomeruli. Hematoxylin and eosin grading was less successful in the kidneys than in the lungs. However, it was of definite value, matching closely with Oil-Red-O grading in all but a few instances.

Brain sections containing fat were seldom found; hence grading was not carried out. It was considered more appropriate to describe the occasional, positive sections as having minimal, slight, or moderate quantities of fat. Recognition of fat vacuoles in hematoxylin and eosin sections of brain was difficult; therefore, quantification of the fat without examination of Oil-Red-O stains was unreliable.

Independently of the microscopic study, the abstracts of the clinical records were combed, giving special attention to duration of life, type and distribution of wounds, presence or absence of fractures, degree of shock, and prominence of cardiovascular, pulmonary, renal and cerebral symptoms. Other pathologic findings at autopsy, especially those referable to the cardiovascular system and lungs, were tabulated in an attempt to discover if their presence could be related statistically to the amount of fat embolism observed microscopically.

Results of Analysis

General Aspects of Cases. The ages of the patients ranged from 18 to 32 years. The average age was 22; and less than 10 per cent of the patients were over 25. Shell fragments caused wounds in 85 per cent of the injured soldiers, while 15 per cent were caused by bullets. Ninety per cent of the shell-fragment wounds and 33 per cent of the bullet wounds were multiple. Seventy-five per cent of the patients had fractures. In a few instances, significant burns complicated the injuries. The anatomic distribution of the wounds is presented in Table 1.

Table 1. Distribution of Wounds in 110 Cases

Location	Number of Cases
Head and neck	4
Trunk	22
Mixed, excluding extremities	5
Extremities	20
Mixed, including extremities	59

The duration of life after injury ranged from seconds to 28 days. Approximately 25 per cent of the patients expired in less than 24 hours; 40 per cent survived between 1 day and 1 week; and 33 per cent died later than 1 week following injury.

Incidence. Of the 89 patients of whom Oil-Red-O sections of lung were examined, 83 (93 per cent) showed pulmonary fat emboli. Using intravascular vacuolation in routine hematoxylin and eosin sections as the criterion for fat embolism, findings were positive in 87 out of 110 cases, an incidence of 79 per cent. It is apparent that minimal degrees of embolism occasionally escaped detection when fat stains were not used. Analysis of both routine and fat stains, as presented in the section on Materials and Methods, revealed that 50 per cent of the soldiers showed no fat embolism or only minimal degrees of it (Grades 0 to 2); 31 per cent showed slight degrees (Grades 3 and 4); and 19 per cent showed moderate to marked degrees (Grades 5 to 7).

Extensive experience with both routine and fat stains has shown that systemic fat emboli are most abundant and easiest to recognize microscopically in the kidneys. For this reason, the presence of fat in the kidneys has been regarded as the most sensitive pathologic index of the entrance of emboli into the systemic circulation. Previous studies by workers in this laboratory,^{9 13} and by others,¹⁴ have revealed that (1) renal involvement rises in incidence and grade with increasing degrees of pulmonary fat-embolism and (2) minimal to slight degrees (Grades 0 to 4) of the pulmonary fat-embolism are only rarely accompanied by the appearance of fat in the kidneys.

For the two reasons stated, renal fat stains were performed routinely in the present series only in cases in which the grade of pulmonary fat-embolism was 4 or greater. In 26 such instances, the kidneys were positive in 17. The grade of pulmonary fat-embolism in the remaining cases being 3 or less and hematoxylin and eosin sections of the kidneys disclosing no evidence of fat embolism, it was considered justifiable and conservative to grade the renal fat-embolism as "none to minimal" (Grades 0 to 2). Combining the results of grading both routine and fat stains, it was found that 92 per cent of the 110 patients showed none or minimal renal fat-embolism. Four per cent exhibited a slight degree (Grades 3 and 4), and an additional 4 per cent showed moderate to marked degrees (Grades 5 to 7).

Because of the importance of cerebral fat-embolism as a potential cause of death, sections of the central nervous system were studied with fat stains whenever the pulmonary grade was 4 or greater and brain tissue was available. Usually one section of cortex was examined. Often additional sections from cortex and other parts of the brain were studied as well. In the 24 cases in which the brain was stained with Oil-Red-O, findings were positive in five. Cerebral fat-embolism was less common and almost always less marked in degree than renal fat-embolism. Of the 24 cases mentioned previously, the kidney was positive for fat in 15, an incidence three

times that of the brain. Similarly, in a previous series dealing with various types of trauma, the kidney was positive for emboli in 18 and the brain in only 9 of 42 cases in which both renal and cerebral fat stains were made.¹³ In neither the present nor a previous series was the brain positive when the kidney was negative. In three of four cases of the present series in which renal fat-embolism was slight, cerebral embolism was minimal in two and absent in the third (in the fourth case no Oil-Red-O section of brain was available). In three of four cases in which renal fat-embolism was moderate to marked, brain embolism was minimal in two and moderate in the third (in the fourth case no Oil-Red-O section of the brain was available).

In summary, about 90 per cent of the soldiers dying in hospitals less than 1 month after battle trauma showed pulmonary fat-embolism at autopsy; however, only 19 per cent of them showed more than a slight degree. Less than 10 per cent of the soldiers had more than minimal renal fat-embolism; and a much smaller percentage had more than minimal cerebral involvement.

With regard to the comparative incidence and severity of fat embolism in various types of trauma, our experience has been that the highest incidence and severest grades were observed in individuals who had been severely beaten and had extensive contusions of the extremities. There were 13 such cases. A study of more than 50 deaths following vehicular accidents revealed an incidence and severity of fat embolism roughly paralleling that complicating battle trauma. The lowest incidence and mildest degrees of pulmonary fat-embolism among the large groups of trauma cases studied were in patients who died of single bullet wounds of the head or chest (over 40 cases examined).

Although an organized study was not made of the incidence of pulmonary fat-embolism in nontrauma cases, it has been impressive whenever present because of its rarity and its mildness.

In 18 cases of patients dying after extensive burns, findings showed that pulmonary fat-embolism was absent or minimal in 15, with slight embolism in two cases and moderate embolism in the other case.

Pathogenesis. Little new information on the pathogenesis of fat embolism was obtained from an analysis of the present series of cases. It was not possible to make an accurate estimate of the amount of soft-tissue and bone injury from the autopsy records; hence these injuries could not be related to the degree of pulmonary fat-embolism. However, it was feasible to make a correlation between the topography of the wounds, the presence or absence of fractures, and the amount of fat in the lungs.

It was found that only one patient showed moderate pulmonary fat-embolism among the 31 patients having wounds confined to the

head, neck, trunk, or a combination of these areas. In contrast, 20 of the 79 patients whose extremities were involved showed moderate to marked embolism. One explanation for this difference is probably the larger amount of osseous damage suffered in the extremity cases. Of the 79 extremity cases, 63 were associated with fractures and 19 of these showed moderate to marked pulmonary fat-embolism. On the other hand, only one of the 16 patients not having fractures exhibited moderate embolism.

Although the wounded-in-action cases provided no opportunity for correlation between the amount of soft-tissue damage and the degree of fat embolism, study of the 13 cases of death due to beating suggested the importance of trauma to adipose tissue in the production of fat embolism. At autopsy, these patients commonly showed marked pulmonary fat-embolism and occasionally a marked systemic fat-embolism, despite the absence of obvious fractures. It is possible, of course, that jarring of the bones with microscopic fractures provided a source of embolic fat in these cases.

Clinical Aspects. An attempt to delineate a pulmonary symptom complex in the present series of cases was unsuccessful. Dyspnea and cyanosis were often mentioned as prominent features in the courses of these patients. However, there appeared to be no correlation between these symptoms and the amount of fat in the lungs. An occasional patient had a more or less acute onset of respiratory distress at some interval after injury and died, showing moderate to marked pulmonary fat-embolism. On the other hand, such histories were also obtained in association with only minor degrees of embolism, and were usually absent in cases showing high grades.

In this series, there was no evidence to support a cardiovascular symptom complex. No signs were observed of right-sided heart failure which could be correlated with moderate or marked pulmonary fat-embolism. In a small percentage of the cases in which it was possible to estimate the degree of shock, no correlation was demonstrable between it and the pulmonary fat content. Furthermore, the occasional occurrence of unexpected sudden death bore no consistent relationship to the quantity of embolic fat in the lungs.

With reference to systemic fat-embolism, ophthalmoscopic examinations and laboratory examinations of the urine for fat were not recorded in our abstracts. Cutaneous petechiae were noted in occasional cases of the series, but could not be correlated statistically with the degree of fat embolism at autopsy.

Only two cases presented a clinical picture suggestive of cerebral fat-embolism. At autopsy, one had a moderate degree, while the other showed cerebral edema but no fat-embolism.

Case 1 (Cerebral Fat-Embolism). A 29-year-old Korean soldier was admitted to an army surgical hospital 7 hours after being treated at a battalion aid station. On arrival, he was in shock and had a

blood pressure of 76 systolic, 48 diastolic. He had traumatic amputations of the right leg through its lowest third, of the left hand, and of several fingers of the right hand. There were open, comminuted fractures of the left tibia and fibula. Multiple shell-fragment wounds of the right hand and scalp were present. Both corneas showed abrasions.

Several pints of blood were administered, and 2 hours later the patient was taken to the operating room. A guillotine amputation of the right lower leg was performed through its lowest third. A similar amputation of the lower left forearm was performed. The first, second, third, and fourth fingers of the right hand were amputated through the proximal phalanges. Débridement of the left leg, foot, ankle, the right hand, and the scalp was carried out.

The patient's immediate postoperative course was not unusual. However, 24 hours following operation he became stuporous, and an intracranial lesion was suspected. He was evacuated to another surgical hospital for consultation and, on arrival, he showed respiratory difficulty. His blood pressure was elevated. He was taken to the operating room 35 hours after admission to the battalion aid station and a cranial exploration revealed the brain to be under a considerable amount of pressure. No epidural or subdural hemorrhage was found. Postoperatively, the patient's respirations were labored. His blood pressure dropped and he expired in 10 hours.

At autopsy, the principal gross findings were moderate dilatation of the left ventricle, slight to moderate pulmonary congestion and edema (lung weight: 600 gm. each), cerebral swelling, and a cerebellar pressure cone. Microscopically, the lungs showed moderate to marked congestion and edema and Grade 6 fat embolism. There were scattered foci of early bronchopneumonia. The kidneys showed Grade 6 fat embolism. Groups of capillaries here and there in the myocardium were plugged with fat. The brain exhibited groups of capillaries filled with fat in all sections, including cerebral cortex, basal ganglia, brain stem and cerebellum. Small foci of rarefaction surrounded some of the emboli in the cortex and basal ganglia. Occasional vacuoles produced by fat emboli were seen in the splenic and adrenal sinusoids in hematoxylin and eosin sections.

Case 2 (Cerebral Fat-Embolism). A young Korean soldier was injured by a land-mine explosion sustaining traumatic amputation of the lowest third of the right leg, multiple shell-fragment wounds of the left leg with avulsion of the skin of the knee, shell-fragment wounds of the hands and of the left upper inner arm with an axillary hematoma, and avulsion of the scrotum with fragmentation of the left testis.

On arrival at a Marine medical battalion aid station, his blood pressure was 75 systolic and 65 diastolic, pulse 120, and respirations 20. His chest was clear to percussion and auscultation. There was

generalized abdominal tenderness. Under gas-oxygen-ether anesthesia, a guillotine amputation of the right lower leg was made; the left testis was excised, and the multiple wounds were débrided. Six pints of blood were given before and during the operation.

Postoperatively, the patient sank into deep coma. On the first postoperative day, his blood pressure was 120 systolic and 60 diastolic, pulse 110, and respirations 20. His chest was clear; and his abdomen was slightly rigid. Catheter output of urine was 1 liter. Early during the second day his temperature was 102° F. He had noisy respirations due to retained secretions; otherwise there was no change in his condition. About 49 hours following operation and 57 hours after injury, he developed signs of an expanding intracranial lesion. The right pupil was larger than the left. He had weak, generalized "shaking" convulsions, and his blood pressure rose to 170 systolic, 100 diastolic. A tracheotomy was performed. Bilateral cranial exploration was negative. Skin petechiae appeared. Death occurred 6 hours after the onset of the final episode, *i. e.*, 55 hours after the first operation.

The most striking finding at gross autopsy was the presence of petechiae disseminated throughout the white matter of the brain. These were most numerous in the region of the substantia nigra. The gray matter appeared uninvolved. Microscopically, the brain showed numerous round to oval areas of infarction; some of the larger ones exceeded in dimension the diameter of a low-power field. The infarcts were characterized by disintegration of the intercellular framework, decrease of its tinctorial properties, nuclear shrinkage and, in some instances, infiltration of polymorphonuclear leukocytes. Occasional petechiae were seen; but these were less numerous than the anemic infarcts. The latter were distributed chiefly in the cortical white matter, but they were also present in the deeper layers of the cortical gray matter and elsewhere. Oil-Red-O stains revealed that many of the capillaries and small arteries, especially in the gray matter of the cortex, were distended by fat globules. Only occasional infarcts in the white matter contained demonstrable fat at their centers.

Sections of the lungs revealed Grade 7 embolism and numerous foci of bronchopneumonia. The heart exhibited fat emboli distending groups of small vessels. The kidney showed a Grade 5 embolism and moderate numbers of tubular casts of hemoglobin type. In the sinusoids and portal space vessels of the liver, there were scattered vacuoles produced by fat emboli. The parenchymal cells in the central halves of the lobules contained numerous medium-sized fat droplets. The remainder of the organs exhibited occasional intravascular vacuoles suggesting fat emboli.

Pathology. Results of this study did not reveal any constantly occurring gross pathologic changes in lungs showing fat embolism. Neither edema, focal hemorrhage, patchy emphysema, nor atelectasis

Table 2. Average Combined Lung Weights in Fat Embolism

Grade of Embolism	Time of Death			
	Less Than 1 Day	From 1 to 2.5 Days	From 2.5 to 5 Days	More Than 5 Days
0-2	1246 (12 cases)	963 (4)	1300 (8)	1171 (29)
3-4	1404 (7)	1020 (2)	1064 (8)	1135 (14)
5-7	871 (5)	931 (4)	1235 (3)	1058 (6)

were described consistently in the lungs with the severer grades of embolism. Table 2 shows the average combined weights of 102 pairs of lungs correlated with the grades of fat embolism at various times at which death occurred following injury. This table obviously affords no support for the thesis that fat embolism favors the development of pulmonary edema.

After considerable experience, it became possible to recognize with ease the vacuoles produced by fat emboli in hematoxylin and eosin sections of the lung. In the larger vessels these appeared as large, round or oval spaces which often coalesced (Fig. 1). Unlike arte-

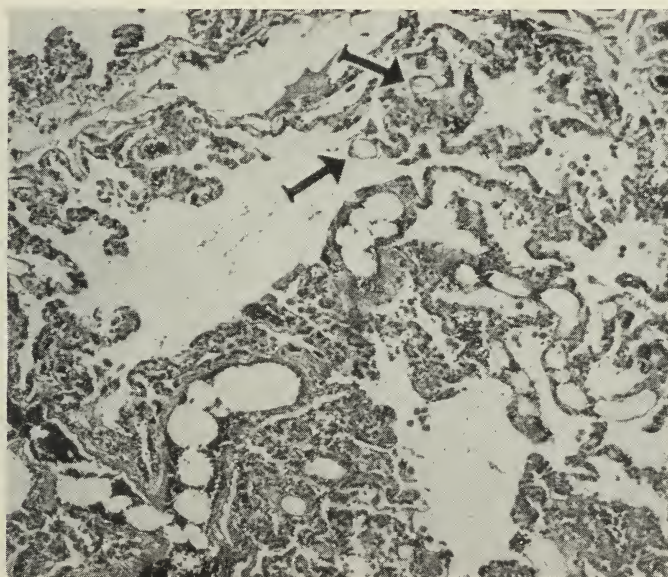


FIGURE 1. Lung. The arteries contain multiple round and oval vacuoles. Several alveolar wall capillaries are distended by large vacuoles. (X100 H & E)

factual spaces which merely displaced intravascular red cells, the vacuoles of fat compressed them so that their surfaces became concave along the margins of the vacuoles. Arterioles and capillaries were plugged and distended by round, oval or reniform vacuoles. At the periphery, the vessel walls and their endothelial linings were tightly compressed suggesting great tension (*see* Fig. 1). In sections of tissues fixed in acid formalin, the deposition of delicate, golden-yellow crystals of formalin pigment in the vacuoles was of confirmatory value in identifying them as fat.

In 4 of the 110 cases, bone-marrow emboli were found in small numbers in the pulmonary arteries (Fig. 2). A tiny fragment of embolic skin was seen in one case. It was common for thrombi composed largely of fibrin and eventually penetrated by endothelial cells to lay in juxtaposition to the embolic fat globules. Since more often than not these thrombi were situated away from the fat emboli, it seemed probable that the association of the two was of a collision type.

The use of fat stains revealed a striking picture, often outlining the pulmonary arterial tree and alveolar wall capillaries (Fig. 3).

The topographic localization of fat emboli within the lungs was not worked out in detail. Sections taken from various regions of the lungs generally contained fairly similar amounts of fat. Occasionally, one section of lung differed by two grades from another, and rarely by three. In sections in which only a small amount of fat was present, it was often confined to a thin rim of lung beneath the pleural surface.



FIGURE 2. Lung. Bone marrow embolus in artery. (X200 H & E)



FIGURE 3. Lung. Arteries and alveolar wall capillaries are plugged with fat. The alveolar walls present a beaded appearance. (X100 Oil-Red-O)

Microscopically, just as grossly, it was not possible to correlate the presence of pulmonary fat-emboli with focal edema, hemorrhage, emphysema, or atelectasis. Table 3 shows the relations of pulmonary fat-embolism to the time of death following injury.

Table 3. Relation of Fat Embolism to Duration of Life After Injury

Category	Time of Death				
	Less Than 1 Day	From 1 to 4.0 Days	From 4.1 to 7 Days	From 7.1 to 14 Days	From 14.1 to 30 Days
Number of cases	26	24	22	26	9.0
Average grade	2.5	3.4	2.9	2.2	2.0
Per cent of cases moderate to marked	19%	33%	23%	12%	0

The severest degrees of fat-embolism were seen most often in the first week after injury, the peak incidence being in the early part of the week after the first day. There was a definite tendency for the pulmonary fat-content to decrease during the second, third, and fourth weeks. At one extreme, fat embolism was seen in patients who presumably lived no longer than minutes; at the other, in the wounded-in-action series, it was demonstrated as late as the 24th day following injury.

No clues were apparent from the histologic sections of lung as to how the embolic fat was eliminated. It was unusual to be able to demonstrate fat droplets within the alveoli. Occasionally, they were extravasated with blood. In one case, there was focal massive outpouring of fat into the air spaces in the absence of obvious hemorrhage. It has not been possible to recognize phagocytosis of extravasated fat by alveolar phagocytes. The lipochrome granules, so commonly seen in these cells, stained an orange or rust color with Oil-Red-O in contrast to the bright red of the embolic fat. Likewise, the quantity of phagocytes bore no consistent relationship to the amount of fat within nearby vessels. Only one case showed a reaction of vascular endothelium to fat emboli. In this instance, the endothelial cells surrounding the fat were filled with numerous small, uniform vacuoles, presumably fatty in nature. No variation in the appearance of the emboli was detected in relation to time. The embolic fat seen in several cases in which death occurred less than 10 hours after injury appeared identical to that observed within pulmonary vessels as late as 24 days after injury. It is possible that local enzyme activity is responsible for the gradual elimination of the pulmonary fat or that the droplets are being propelled continually into the systemic circula-

tion and excreted by the kidneys. The failure to find fat regularly in the air spaces does not exclude the possibility of rapid elimination in the sputum.

When more than a minimal amount, usually renal fat-embolism was easy to recognize in hematoxylin and eosin sections by observing the vacuolation of the glomerular tuft capillaries (Fig. 4). When the degree of embolism was minor, the fat was most apt to appear in the glomeruli near the renal capsule. Emboli were seen also in the inter-tubular vessels, chiefly in the cortex (Fig. 5). Rarely was fat visible in the form of droplets or casts within the tubules (Fig. 6). In a single case of death due to beating, fibrin thrombi lay in juxtaposition

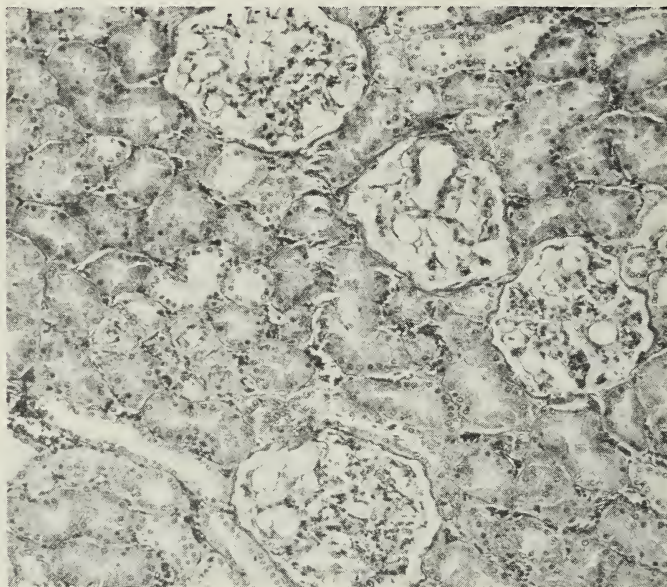


FIGURE 4. Kidney. Many glomerular tuft capillaries are distended by fat vacuoles. (X100 H & E)

to the fat emboli in the glomeruli. Fat-embolism was seen in the kidneys as early as 10 to 18 hours after injury and as late as 17 days.

Emboli were scattered in groups in both gray and white matter of the central nervous system. Vacuolation in hematoxylin and eosin sections of brain was often difficult to recognize. Occasionally, an empty, dilated vessel with a tenuous wall was highly suggestive of fat-embolism (Fig. 7). Parenchymal lesions secondary to the emboli were observed in only two cases. In these two cases, petechiae and small areas of anemic necrosis appeared (Fig. 8), especially in the white matter of the cortex, but elsewhere as well. The infarcts were characterized by a loosening and loss of tinctorial properties of the intercellular framework and shrinkage of both nerve and glial cells.

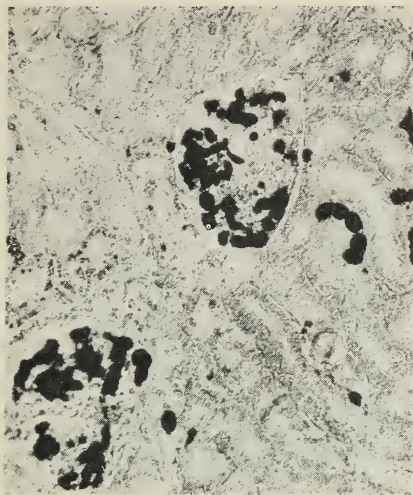


FIGURE 5. Kidney. The glomeruli contain pretzel-like masses of fat. The interlobular vessels are also plugged with fat. (X100 Oil-Red-O)

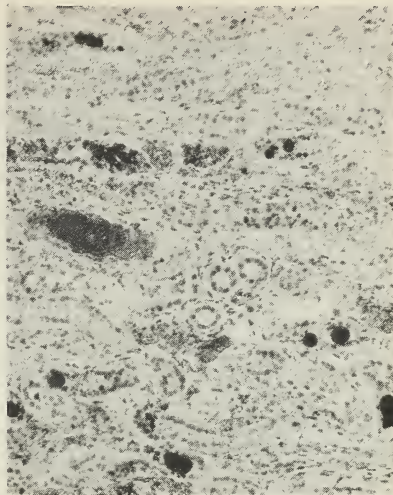


FIGURE 6. Kidney. The uniformly black intratubular casts are fat. The paler granular casts are of hemoglobin type. (X100 Oil-Red-O)

Affinity was decreased for both eosin and fat stains. Some lesions showed infiltration by polymorphonuclear leukocytes. In occasional infarcts, centrally situated capillaries or arterioles were plugged with fat.

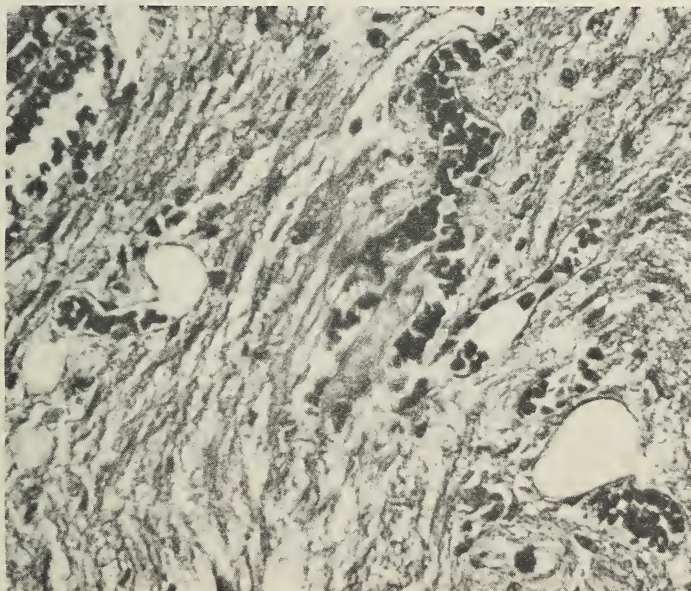


FIGURE 7. Posterior Pituitary. Three small vessels are distended by vacuoles suggestive of fat. (X650 H & E)

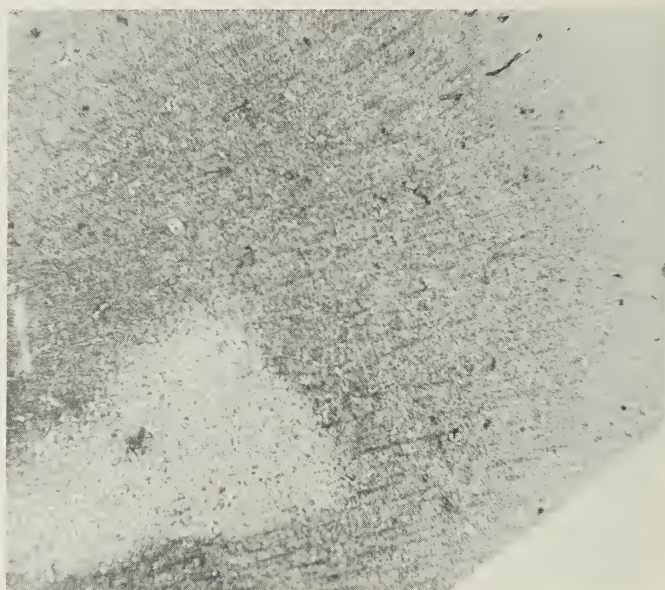


FIGURE 8. Cerebral Cortex. A pale, irregular area of rarefaction is seen at the junction of gray and white matter. Small black streaks, representing fat emboli in the gray matter can barely be made out at this power. (X50 Oil-Red-O)

Using fat stains, emboli were seen most commonly in the gray matter of the cortex (Fig. 9). A confusing element in the interpretation of fat stains of the brain was the frequent presence of phagocytes containing lipochrome granules in a perivascular location. These granules, which had a golden color in hematoxylin and eosin sections, seemed to be entirely unrelated to fat emboli; and often they appeared in their absence.

Oil-Red-O stains were made on only a few heart sections. In Case 1, small groups of capillaries were distended with fat globules (Fig. 10). The myocardium showed a few scattered small foci of degeneration characterized by hyalinization of fiber cytoplasm and nuclear pyknosis. It was not certain that these changes were ante-mortem. Fatty degeneration of muscle fibers surrounding the emboli and petechiae were not observed.

Next to the lungs and kidneys, fat emboli were most easily recognized in hematoxylin and eosin sections of the adrenals and spleen. Here they produced characteristic round, oval, and sausage-shaped vacuoles which were most numerous in the subcapsular zones (Figs. 11 and 12). In occasional cases, vacuoles highly suggestive of fat emboli were seen in other organs. With the exception of the brain, degenerative parenchymal lesions secondary to fat embolism were not observed.

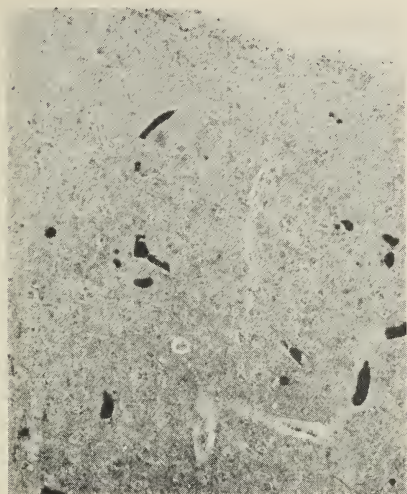


FIGURE 9. Cerebral Cortex. The gray matter contains numerous fat emboli. (X100 Oil-Red-O)



FIGURE 10. Myocardium. A group of capillaries contain fat emboli. (X108 Oil-Red-O)



FIGURE 11. Adrenal Cortex. Many sinusoids are distended by round and sausage-shaped vacuoles produced by fat emboli. (X138 H & E)

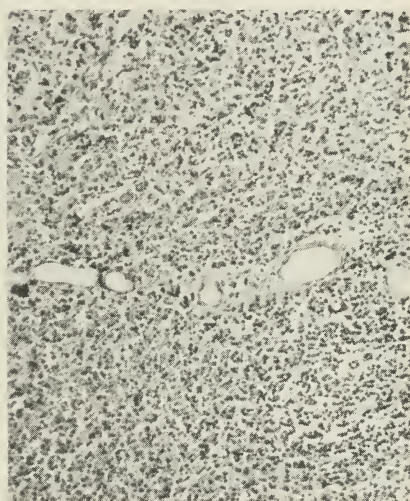


FIGURE 12. Spleen. Several arterioles are distended by vacuoles of fat. (X100 H & E)

Discussion

Because of the many variables existing in any large series of trauma cases, it is not reasonable to compare the high incidence of fat embolism in our series (about 90 per cent) with incidences in most other reported series. Mallory's collection of autopsies on World

War II wounded¹⁰ is perhaps as comparable to our series as any in the literature. His incidence of pulmonary fat-embolism (67 per cent) is lower than ours. However, his figure for "significant" embolism (20 per cent) tallies closely with this series for moderate to marked embolism (19 per cent). Likewise, our incidence of moderate to marked systemic fat-embolism (4 per cent) is identical to that given by Mallory for "significant" systemic fat-embolism.

This series does not support the widely accepted concept of fat embolism as a clinicopathologic entity in regard to its role in producing significant pulmonary dysfunction. Analysis of our cases did not reveal statistical evidence that fat embolism causes pulmonary distress. Likewise, there was no pathologic correlation between the amount of pulmonary fat-embolism observed and the presence and degree of pulmonary edema, pulmonary hemorrhage, atelectasis, emphysema, or right-sided cardiac dilatation. Mallory was similarly unable to correlate fat embolism and pulmonary edema on a statistical basis.¹⁰ It might be possible that serious pulmonary embarrassment might result from the presence of embolic fat in an occasional individual case. However, this finding must be quite uncommon, or it would be detected in some of the large series investigated, such as this series.

In reviewing the literature on fat embolism, the investigators were impressed by the lack of "controls," *i. e.*, injured patients who died showing none or, at most, slight fat embolism at autopsy. The failure to compare such cases with cases of moderate to marked fat embolism exposed one to the risk of "*post hoc, ergo propter hoc*" fallacious reasoning in implicating fat embolism as the cause of various clinical and pathologic findings. Furthermore, the frequent absence of pulmonary symptoms and signs antedating the onset of cerebral fat-embolism^{7 11 18} is difficult to explain if one holds that pulmonary fat-embolism produces respiratory embarrassment. Cerebral fat-embolism, with possible rare exceptions, is encountered only when the degree of pulmonary involvement is moderate to severe.

Our conclusions, based on clinical data, are identical to those of Armin and Grant who employed experimental methods.¹ These authors produced pulmonary fat-embolism by injecting rabbit perirenal fat into ear veins of unanesthetized rabbits. They estimated the amount of fat necessary to inject in order to produce a degree of pulmonary fat-embolism equivalent to a severe degree in the human. This estimate was based on measurement of the extractable fat content of severely involved human and rabbit lungs, as well as on grading of histologic sections.

From their experiments, Armin and Grant reported four conclusions. (1) The production of pulmonary fat-embolism corresponding to a severe grade in the human caused no change in blood pressure,

pulse rate, or respiration in the normal rabbit. The dose given to produce such an embolism was much less than that required to kill the rabbit. (2) In animals subjected to severe hemorrhage (averaging about 40 per cent of the blood volume), the additional production of severe, pulmonary fat-embolism did not alter the clinical picture or increase the mortality above that due to hemorrhage alone. (3) The production of severe, pulmonary fat-embolism had no effect on the response of the animals to transfusion after hemorrhage. (4) Unless the rabbit tolerates fat embolism better than man, human gross pulmonary fat-embolism is unlikely to provoke symptoms or to be solely or in part responsible for death after injury.

As a corollary to our inability to relate evidence of pulmonary distress to fat embolism, the percentage of trauma deaths attributable to fat embolism was exceedingly low as compared with rates given elsewhere in the literature (in the vicinity of 1 per cent). Cases listed as fatal were only those in which death was associated with a typical cerebral syndrome clinically and a significant degree of cerebral fat-embolism pathologically. The high mortality rates given in the literature depend largely upon the assumption that moderate to severe grades of pulmonary fat-embolism, whenever demonstrated, represent a primary cause of death or are a major factor contributing to death.

Several objections may be raised to the nature of the material from which our conclusions have been drawn. (1) The clinical abstracts at our disposal were not always detailed. (2) In caring for large numbers of wounded soldiers most of whom were seriously ill, field surgeons may not have accurately or uniformly recorded any but the severest signs or symptoms. (3) Pathologically, it was impossible to judge the ages of pulmonary fat-emboli; *i. e.*, it could not be determined in any one case whether the emboli had accumulated in successive crops over a period of time or whether they had involved the lungs in a single, massive onslaught. Such a determination, if possible, would undoubtedly have increased the value of our clinicopathologic correlation. Despite these possible objections (one or more of which applies equally to the other series in the literature), it is believed: (1) that little in the way of conclusive proof has been offered for the opinion that moderate to severe pulmonary fat-embolism causes significant pulmonary dysfunction or death, and (2) that other possible sequelae of injury have not been adequately excluded as causative agents of pulmonary signs and symptoms when such appear.

It was evident that cerebral fat-embolism was seldom present in significant amounts, since only 1 per cent of the cases of battle trauma coming to autopsy showed this entity.

Conclusions

A clinicopathologic analysis of 110 cases of death occurring at military hospitals up to 4 weeks after battle trauma was made in order to determine the incidence and significance of fat embolism.

Fat embolism, as evidenced by the presence of fat droplets in the pulmonary vessels, was demonstrable in approximately 90 per cent of the 110 patients. In only 19 per cent was the degree of pulmonary fat-embolism more than slight.

Of the 110 patients, only 4 per cent showed more than slight systemic fat-embolism, as evidenced by the appearance of fat in the kidneys; and only 1 per cent showed fatal (cerebral) fat embolism.

From both clinical and pathologic viewpoints, there was no evidence from the analysis that the presence of fat in the lungs causes pulmonary dysfunction or death. This finding is at marked variance with the concepts of many writers concerning the significance of pulmonary fat-embolism.

Fat emboli may be recognized and quantitated with considerable accuracy without the use of fat stains by searching for and estimating the amount of intravascular vacuolation. This can be done with greatest success in the lungs, and with somewhat less success in the kidneys.

References

1. Armin, J., and Grant, R. T.: Some Observations on Gross Pulmonary Fat Embolism. Cited by Grant, R. T.: *Brit. M. Bull.* 10: 17, 1954.
2. Artz, C. P.: Personal communication.
3. Darrach, W.: "Discussion of a Case Report." Harris, R. I.; Perrett, T. S.; and MacLachlin, A.: *Fat Embolism*, p. 1113. *Ann. Surg.* 110: 1095, 1939.
4. Denman, F. R., and Gragg, L.: *Fat Embolism: A Diagnostic Enigma.* *Arch. Surg.* 57: 325, 1948.
5. Glas, W. W.; Grekin, T. C.; and Musselman, M. M.: *Fat Embolism.* *Am. J. Surg.* 85: 363, 1953.
6. Groendahl, N. B.: Cited by Scuderi, C. S.: *Fat Embolism: A Clinical and Experimental Study.* *Surg. Gynec. & Obst.* 72: 732, 1941.
7. Groskloss, H. H.: *Fat Embolism.* *Yale J. Biol. & Med.* 8: 59, 175, and 297, 1935-1936.
8. James, E. S.: *Fat Embolism.* *Canada M. Assn. J.* 62: 548, 1950.
9. Lukeman, J.: *Fat Embolism.* Annual Historical Report, 406th Medical General Laboratory, pp. 92-94. United States Army, Washington, D. C., 1951.
10. Mallory, T. B.: "General Pathology of Traumatic Shock"; *The Physiologic Effects of Wounds*, pp. 283-305. Board for the Study of the Severely Wounded, North African-Mediterranean Theater of Operations. Department of the Army, Office of The Surgeon General, Washington, D. C., 1952.
11. McArdle, M. J. F.: Cited by Newman, P. H.: *The Clinical Diagnosis of Fat Embolism.* *J. Bone & Joint Surg.* 30B: 290, 1948.
12. Robb-Smith, A. H. T.: *Pulmonary Fat Embolism.* *Lancet* 1: 135, 1941.
13. Scully, R. E.: *Fat Embolism.* Annual Historical Report, 406th Medical General Laboratory, pp. 197-201. United States Army, Washington, D. C., 1952.
14. Vance, B. M.: *The Significance of Fat Embolism.* *Arch. Surg.* 23: 426, 1931.
15. Warthin, A. S.: *Traumatic Lipaemia and Fat Embolism.* *Internat. Clin.* 4: 171, 1913.
16. Whitson, R. O.: *A Critique of Fat Embolism.* *J. Bone & Joint Surg.* 33: 447, 1951.
17. Wilson, J. V., and Salisbury, C. V.: *Fat Embolism in War Surgery.* *Brit. J. Surg.* 31: 384, 1943-1944.
18. Winkelman, N. W.: *Cerebral Fat Embolism.* *Arch. Neurol. & Psych.* 47: 57, 1942.

Chapter 23

Future Research in Support of the Battle Casualty

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The responsibility to problems of the combat casualty is not limited to the military surgeon, nor is it limited in time to the period of combat. It is a continuing, national responsibility with ever-increasing overtones to the civilian as well as to the military population.

The problems observed in the care of the battle casualty center around two considerations: the local destructiveness of the wounding agents, and the effect of the wound on the entire body. Thus there are two major effects of the trauma: the local tissue injury, and the subsequent involvement of the circulatory system. These might be considered two sites of primary injury and they are the two which may produce a continuing, deleterious influence. The third site of injurious action, which may be primary and continuous, is that of the nervous system. The possible role of this injury in producing a deleterious effect is less obvious and needs further study.

Research in support of the battle casualty must therefore proceed in several directions in order to include the problems of tissue destruction, hemorrhage, resuscitative agents, treatment, and the sequelae of injury.

As suggested above, research must proceed not only at the national level but, should the occasion arise, in the combat theater.

Research at the National Level

The Local Wound

This, the focal point of the entire insult, has not been adequately studied. Continued investigation of wounding agents and of wound ballistics is required.

Knowledge is needed as to the extravasation of fluid, blood, albumin, globulin, and plasma expander into the wounded tissue. The extent and duration of the extravasation are unknown.

The changes in the circulation in the wounded extremity should be studied as to the degree and duration of these changes. This study might provide a sound background for the continued study of the absorption of endogenous toxins and of the electrolyte shifts occurring in traumatized tissue.

The delineation of viable from nonviable tissue at the time of primary débridement is a very practical problem of tissue conservation which requires additional study.

Wound infections seldom appeared to be a major, life-endangering problem under military conditions in Korea. However, the ever-present possibility of mass casualties, inadequate surgical therapy, as well as the inevitable loss of tissue and function, and the increase in hospital days secondary to noncritical wound infections require that studies of wound infections be continued.

Since the achievement of a healed wound is a primary objective in the case of the injured, wound healing and the factors affecting it in the battle casualty deserve continued study. It was the clinical experience in Korea that wound complications were among the major causes of death in casualties with posttraumatic renal insufficiency. The underlying defects, therapeutic or biologic, require elucidation.

The Circulation

The study of factors controlling the circulation should be expanded at all echelons. This cannot be overemphasized. At the practical level, the problems lie in the support of the circulation after hemorrhage has been controlled and transfusion instituted. Study should emphasize not only the factors controlling the circulation, but also the effect on the circulation of analgesic and anesthetic agents.

The observations already made in Korea are threefold. (1) Massive transfusions were required to maintain the blood pressure and prevent death after very severe injuries. (2) Vascular collapse was more frequent following cranial and abdominal trauma, but it also occurred following wounds limited to the extremities. (3) Post-traumatic renal insufficiency was a major complication among battle casualties.

Abdominal Wounds

Although the mortality from abdominal injury has steadily decreased, it remained at 12 per cent in the experiences reported from Korea. Studies, both at the basic and the clinical level, should be made in an effort to reduce this mortality. Particular attention should be paid to the control of intra-abdominal hemorrhage, peritonitis, and of wounds of the liver, colon, kidney, urethra, duodenum and inferior vena cava.

Injuries of the Brain and Spinal Cord

Although the figures from Korea are not available, the mortality of casualties with injuries of the central nervous system reaching the hospital probably ranged from 15 to 25 per cent. If patients with injuries of the brain reached the hospital alive, it would seem that

means could be found to prevent the progression of the injury short of fatality.

Burns and Irradiation

These were not major problems in Korea and are included only because of the obvious increase in destructiveness of military weapons.

Plasma Expanders and Red Blood-Cell Preservation

Continued study should be made to increase the effectiveness of plasma volume expanders. Although the expanders used in Korea (dextran and modified fluid gelatin) were reasonably satisfactory under the conditions of a 3.5-hour period of evacuation, they might not prove effective should the time lag increase. An expander is needed which will stay in the blood stream longer than would those studied in Korea. An even more satisfactory preparation might be achieved by the success of the present studies of the long-term preservation of red blood cells.

The Fate of the Red Blood Cell After Massive Transfusions

Clinical experience in Korea dictated the need for massive transfusions in support of the casualty with extensive injuries. In spite of intensive studies, the fate of the red blood cell was never adequately ascertained.

Anesthesia

Whereas the use of anesthesia is an aid in the correction of the injury to the wound and the injury to the circulatory system by débridement and transfusion, it furthers possible injury to the nervous system. Time and again the casualty with a normal blood pressure was found to become severely, even fatally, hypotensive as anesthesia was induced. Additional efforts should be directed toward understanding and minimizing this trauma by carrying out basic studies in anesthesia. These should include studies of the patient in the "unsteady state," *i. e.*, the injured patient whose circulatory system is partially compensated but which collapses after movement, changes in posture, or anesthesia.

Post-traumatic Renal Insufficiency

Although infrequent, post-traumatic renal insufficiency is a major complication among severely injured battle casualties. Continued study should be directed toward understanding the cause of this syndrome as well as toward increasing the effectiveness of therapy. For the severe stages of post-traumatic renal insufficiency, the case fatality rate remains at least 50 per cent.

Refractoriness to Transfusion

As a fatal complication, this condition develops very infrequently; however, it was the clinical impression in Korea that many of the casualties developed a limited degree of refractoriness as indicated by the need for large transfusions. Although continuation of studies of basic mechanisms is essential, lethal refractoriness was not a frequent complication. It may well be that it is more of a problem in the older patient than in the young battle casualty.

Hepatitis

The author had little personal experience with this complication of transfusion in Korea, but investigators have indicated its importance both in the management of the combat casualty and in civilian patients.

The Systemic Response to Injury

Studies in Korea indicated that the entire body took part in the response to trauma. Among the practical problems encountered which had not been adequately explored were the problems of paralytic ileus and the possible need for support of the autonomic nervous system following severe injury. Although paralytic ileus is observed routinely following abdominal injury, both in military and civilian practice, all too often it is accepted without interest. Like renal vasoconstriction, paralytic ileus may serve as a protective mechanism following injury; but in this day of transfusion and closure of perforated viscera, both responses may add to the morbidity after the initial defects have been adequately corrected. Under conditions of mass casualties, delayed medical attention and inadequate supplies may lead to neglect. Under such circumstances, oral administration of fluids may prove the only feasible means of treating even the seriously wounded. Gastrointestinal absorption of fluids and electrolytes following injury has not been adequately explored.

The autonomic nervous system has not been adequately studied. Its response as to mechanism, degree, duration, and possible deficiencies is an unknown factor in the defense following injury. The role of vasoconstrictors in resuscitation cannot be evaluated adequately until this basic response is elucidated.

The degree, duration and the effect of the adrenal cortical response has been fairly well outlined within the limits of present day methodology. No deficiency in this response was recognized in the battle casualty. Future work should pursue the development of new methods of study with emphasis on fluid and electrolyte shifts within the body. The significance of the decrease in plasma sodium concentration and its therapeutic implications following injury deserves intensive study.

Organ Transplantation

Perhaps the greatest contribution in military medicine might be made in the field of organ transplantation. Work in this sphere should have unlimited support.

Research in the Combat Theater

Should the occasion ever arise again when American troops are engaged in combat, or should the occasion present itself for research work in any combat theater, the opportunity for field research should be immediately grasped. This work should be planned at the present time and organized in such a way that effective work could be instituted at an early date.

Studies in the Division Area

Since approximately 20 per cent of all soldiers wounded by enemy action die before reaching the forward surgical hospital, this should prove the most fruitful site for clinical investigation. Work here should entail not only prevention of injury, but emergency therapy. Much might be accomplished in the field of resuscitation and evacuation by utilizing seasoned clinical investigators at the most forward medical installations as well as at other points throughout the forward chain of evacuation. It is realized that studies in this area would have to be carried out by men who are at the same time physicians and surgeons.

Clinical Surgery and Organized Follow-up

Experiences in neurosurgery and in vascular surgery in Korea demonstrated how much could be accomplished by concentrating on specific clinical problems in the forward area and then selectively evacuating the casualties to specified hospitals in the Communications Zone or in the Zone of Interior. Such work should be planned for every sphere of traumatic surgery. By correlating the experiences in the forward area and in the secondary hospitals, new technics could be developed and immediate follow-up might permit evaluation of a given clinical program or permit the surgeons to revise their program at an early date as experience dictated. The importance of such a program cannot be overemphasized.

The Management and Study of Abdominal Trauma

As described earlier, the mortality from abdominal trauma remains high. In the forward surgical hospital, clinical as well as basic studies should go hand in hand in an effort to describe the cause of fatality while, at the same time, every effort is being made to lower the current fatality rate.

The Study and Management of Patients with Injuries to the Central Nervous System

As with abdominal trauma, this condition is listed separately for added emphasis.

The Management and Study of Casualties with Post-traumatic Renal Insufficiency

Because of the relative frequency of this complication of trauma in the combat theater and because of its high fatality rate, the continued study of renal insufficiency should be established in the combat theater.

Débridement and Wound Infections

Because almost every casualty admitted to the forward hospital has an open wound and some degree of wound infection or contamination, either clinical or subclinical, an unlimited supply of patients is available for the study of the proper management of wounds.

Anesthetic Management

The battle casualty differs from many civilian patients in that, prior to anesthesia, he has lost blood and developed some response of the autonomic nervous system. Since this response appears to be partially blocked by anesthesia, anesthesia is a greater hazard than in many of the nontraumatic casualties. Imaginative leadership in this sphere might well lead to the saving of additional lives and the accumulation of data of fundamental interest.

Study of the Blood and Plasma Expander Program

As in the study of wounds, a study of transfusion should be continued not only because of the inherent problems in such a program, but also because of the tremendous clinical experience which is available for study and analysis.

Burns and Irradiation

Study of these wounds should be carried out if indicated.

Summary

To reiterate, the responsibility to problems of the combat casualty is not limited to the military surgeon, nor is it limited in time to the period of combat. It is a continuing, national responsibility with ever-increasing overtones to the civilian as well as to the military population.

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